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Lewis A. Hurst

SOME DATA ON NATURAL FERTILITY
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Due to changes in organization and of editors, this issue of *Eugenics Quarterly* is being published behind schedule. The September issue will also be delayed, but *Eugenics Quarterly* should be on schedule by the end of this year. We sincerely regret any inconvenience this may have caused our readers.

Vol. 8, No. 2, June, 1961 *Eugenics Quarterly*

APPLICATIONS OF GENETICS IN PSYCHIATRY AND NEUROLOGY*

LEWIS A. HURST

Professor of Psychological Medicine
University of Witwatersrand
Johannesburg, South Africa

RECENT strides in general genetics, biochemical genetics, and human genetics have yielded a rich harvest in the field of psychiatry.

Specific genetic mechanisms underlying important psychoses and several forms of mental defect have been elucidated. This definition of the specific genetic basis of each of our major endogenous psychoses, schizophrenia and manic-depressive psychosis, has provided a firm starting point for the exploration of the interaction between physiodynamic and psychodynamic approaches in these conditions. Moreover, scientifically acceptable data from which to evolve eugenic theory and principles in the field of psychiatry have thus been made available. And finally, evidence that has come to us from the field of biochemical genetics, pointing to enzyme blocks as the process mediating genetically determined pathological processes and states, including those falling within the psychiatric field, gives us a precise vantage point for research and therapeutic attack — most paradoxically in an area in which the central concept (heredity) was formerly equated with irreversibility and prognostic hopelessness.

We shall be in a better position to elaborate these our two major themes of research and therapeutic attack on the one hand, and eugenic policy on the other, when we have cleared the ground by means of presentation of methods and data, to which I now address myself.

METHODS OF INVESTIGATIVE PROCEDURE IN HUMAN GENETICS

The Pedigree or Family History Method

This method is mainly of historical interest and of preliminary suggestive significance. Even before the Mendelian era certain traits or disorders were ob-

*This article is based on one of similar title appearing in the *South African Journal of Laboratory and Clinical Medicine*, Vol. 4, No. 3, Sept. 1958. Our thanks are due to the editor for permission to publish material appearing in that article.

served to "run in families," paving the way for the more effective methods of assessing the fact of heredity and the type of heredity mechanism involved. Sir Ronald Fisher (1956) gave interesting examples of such pedigrees that had pointed the way in his address at a plenary session of the 1956 Congress at Copenhagen. Pedigrees, from the pattern of distribution of affected or unaffected members of the varying degrees of relationship within the family, may provide an indication of whether the trait concerned is transmitted as a Mendelian dominant (transmission in the direct line of descent, a 50% rate of children of one affected parent similarly affected, and descendants of nonaffected members not affected themselves), or as a Mendelian recessive (transmission in the collateral line of descent, a 25% rate of affected children of two overtly normal parents, raised consanguinity rate in parents, and skipping of generations in respect to affected members), or as one of the varieties of sex-linked traits (in the commoner of which, haemophilia, the condition is transmitted by the female and suffered by the male).

Such pedigrees, however, can at most be regarded as of preliminary value, because the *small* size of human families does not statistically warrant the drawing of firm conclusions about the genetic mechanisms involved, and the pedigrees that draw attention to themselves often do so in virtue of their unduly heavy loading (not representative of the average family) with cases exhibiting the trait or disease in question.

From the cue given by pedigrees we are driven on to the more precise and statistically adequate methods which have been called the Contingency Method of Statistical Prediction and the Twin Study Method, the two methods gaining in effectiveness by combination in the Twin Family Method of Kallmann.

The Contingency Method of Statistical Prediction

Before proceeding to the more technical aspect of this method, it should be stressed what effort is made in modern genetic methods to collect a representative, unselected sample. Perusal of Kallmann's (1937) monograph will give an idea of how painstaking such work may be. In the contingency method, large samples of relatives are used, classified by degree of blood relationship to the probands or index cases (carriers of the trait under investigation who form the starting point of enquiry for each family being studied) and considered suitable for comparison with a normal average population group. The technical procedures employed in such an assessment are Weinberg's proband and sibship methods.

The derivation of expectancy figures from the raw data of a particular investigation involves correcting for statistical inequalities in age distribution and in the process of collecting index cases. The most elementary corrective methods used are those of Weinberg. His methods for correcting bias in the selection of cases have been referred to but cannot be elaborated here. It may be instructive, however, to review his abridged method which corrects for inequalities of age distribution. Let us consider a series of relatives being investigated in respect to disease X, which has been found to have a manifestation period ranging from age 20 to age 40. Table 1 shows that although the divisor in calculating the net or uncorrected figure is 300, the divisor in calculating the corrected or expectancy figure is only 150, yielding a result which is twice the net figure.

TABLE 1
METHOD TO CORRECT FOR INEQUALITIES OF AGE DISTRIBUTION

Age Group	All consanguineous persons observed	All cases of Disease X observed (net figure*)	Corrected rate of reference (expectancy figure*)
0-20	100	—	—
20-40	100	10	50
Over 40	100	20	100
No. of persons	300	30	150

*Morbidity Rate: 10% (net or uncorrected): 20% (corrected or expectancy figure).

The reasons for reducing the divisor are as follows: 100 out of 300 cases cannot be counted at all as they are under 20 years of age and thus cannot have reached the minimal age at which the disease can develop. The 100 on the third line in Table 1, denoting the age group 40 and over, all count because they have passed the manifestation period for the disease. The 100 on the second line, denoting those distributed throughout the age period 20-40, may, on the assumption of even distribution throughout this period, be taken as having on the average reached the age of 30, thus having been exposed to only half the risk of developing the condition—so that instead of this figure's contributing a full 100 to the denominator it only contributes 50. Adding our three figures together we arrive at a figure of 150 instead of 300.

More refined methods have been devised by Bernstein, Ilse, Strömgren and Lenz which take cognizance of an uneven or irregular distribution through the risk period. The most precise method employs morbidity statistics analogous to the mortality tables used in life insurance statistics.

The Twin Study Method

This method is based on the fact that monozygotic or identical twins originating from the same fertilized ovum have essentially the same genotypes or hereditary equipment, whereas dizygotic or fraternal twins have no greater chance of having a similar combination of hereditary characters than any pair of ordinary siblings. If, therefore, in a series of twins collected on the basis of the fact that one at least of every pair exhibits the trait or disease under consideration the one-egg twins show a higher proportion of cases in which both members of the twin sets manifest the condition (concordance) than do the two-egg twin sets, we have presumptive evidence of the operation of hereditary factors in the determination of the trait.

With regard to the establishment of the zygosity of twins, i.e., whether the twins are monozygous or dizygous, the Similarity Method (due to Siemens) is now generally used. According to this method twins are classified as monozygotic if they are strikingly similar in general appearance and, in particular, if they are alike in the color of eyes, hair and skin, hair texture, blood groups, facial features, dental irregularities, finger proportions, fingerprints and palm patterns. The presence of reversed asymmetry in handedness, dentition and fingerprints is confirmatory evidence of monozygosity. The foetal-membrane method in its original simplicity is unreliable and is no longer used since monozygotic twins may have separate placentas.

Certain criticisms of inferences from twin studies on the basis of anomalies of development have been raised by Zlotnikoff, Bronson Price, Newman, Dahlberg, N. F. Walker, Waardenburg, Meyer, Curtius and von Verschuer, but the phenomena are too rare to invalidate the main premise involved in twin studies.

Of more general scope is the contention that one-egg twins share a much more similar socioeconomic psychological environment and that higher concordance figures which may be found in this type of twin, as compared with the two-egg variety, may not be due entirely to the greater similarity in genetic equipment. Eliot Slater's (1953*a*) formulation is to this effect:

> It is sometimes assumed that any similarities shown by uni-ovular twins can be attributed to heredity, and differences to the environment, but only the latter half of this asumption can safely be held. From this, however, it does not follow that deductions about the importance of heredity in causing some characteristic cannot be based on the examination of a series of uni-ovular twins, provided that there is no biased selection and that controls, i.e. binovular twins, are available.

Ways of attempting to approach and solve the problem raised are considered in paragraphs numbered 3 and 4 to follow.

The co-twin control method was used by Gesell and Thompson for the study of variations such as those of intelligence produced in selected pairs of monozygotic twins by prearranged differences in training and other environmental influences. As applied to our special problem this method leads to the investigation of whether the concordance rates for the trait or disease in question differ between one-egg sets reared together and those reared apart under different psychological and socioeconomic circumstances. A significant difference in these figures would provide a measure of the contribution of environmental factors to the causation of the trait in question.

The twin-family method of Kallmann includes the combination of the twin aging persons."

and family methods, which permits of the bringing together of certain significant figures and which makes this method a more powerful analytic tool than either of the component methods separately. The main categories of relationship for which morbidity figures are obtained in this method are: one-egg twins, two-egg twins, full siblings, half-siblings, step-siblings, and the general population. This method demonstrates whether or not the individual's chance of developing a given trait increases in direct proportion to the degree of blood-relationship to an index case affected by the trait in question.

A specific example is the best way of conveying the force of this method. The figures for schizophrenia in Kallmann's American study areas follows:

One-egg twins	86.2%
Two-egg twins	14.5%
Siblings	14.2%
Half-siblings	7.1%
Children	16.4%
General Population	0.85%

Inferences from these figures are:

1. The morbidity figures of all degrees of blood-relationship are much higher than in the general population—even in half-siblings the incidence is 8 times greater.

2. With increasing degree of blood-relationship there is increasing degree of morbidity — from 7.1% in half-siblings and 14.2% in full siblings to 86.2% in one-egg twins.

3. The concordance rate for one-egg twins is very high (86.2%) and is approximately 6 times the figure for two-egg twins. Beside this the finding in another part of the study that the concordance of separated one-egg schizophrenic twins is slightly lower than for nonseparated one-egg twins–65.0% as compared

with 71%—appears to imply relatively insignificant environmental factors in causation of the condition.

4. The essential similarity of the figure for siblings and two-egg twins (14.2% and 14.5%) is of intrinsic interest because of postulated lack of significant differences in average genetic equipment. There is the further point that there is an obvious analogy here to the greater similarity of the environment shared by one-egg as compared with two-egg twins: in this case there is a presumption that two-egg twins are on the average likely to share a more common environment than any two siblings taken at random and yet here this environmental factor has exerted no effect on the comparative morbidities.

GENETIC FINDINGS IN PSYCHIATRY AND NEUROLOGY

This section consists of a condensed description of some of the main findings and recent research in the fields of psychiatry and neurology, concluding with a consideration of the condition phenylpyruvic amentia, where something substantial on the biochemical side is already known.

Genetic Findings in Psychiatry

With the review of principles already given, Table 2 should be meaningful. Something may profitably be said in elaboration of the following items.

1. *Manic-Depressive Psychosis.* The genetic mechanism is irregularly dominant with a high degree of penetrance, and with a pronounced tendency to a relatively mild symptomatology in the presence of stabilizing constitutional modifiers. The diagnostic classification should be restricted to cases with acute, self-limited mood swings before the fifth decade of life and without progressive or residual personality disintegration before or after psychotic episodes. So diagnosed, the disease is correlated with a tendency to obesity, cardiovascular disorders, gout and diabetes and high resistance to tuberculosis, and is apparently based on a strictly specific genotype. This condition is genetically unrelated to reactive or neurotic depression, to menopausal and presenile depressions, or to other nonperiodical forms of depressive behavior in the involutional period. The dysfunction may be looked upon as one of regulatory instability, but present information about the biochemical constituent of the underlying genotype, as well as the range of its compensatory adaptiveness, is still far from complete (Kallmann, 1950, 1951, 1953*a*, 1954).

2. *Schizophrenia.* In addition to the figures appearing in Table 2, their implications have been studied in connection with a review of the twin-family method. One additional statistic in relation to the question of dominance or recessiveness of the schizophrenic genotype is that in Kallmann's studies he has found a figure of 16.4% in children with one schizophrenic parent, and 68.1% with both parents schizophrenic. The most cogent view, according to Kallmann, is that the condition is determined by an autosomal and single-recessive unit factor, the penetrance (70%) and phenotypical expressivity of which are limited by polygenic (nonspecific) constitutional modifiers, measurably correlated with the compensatory capacities of the athletic component of physique (Kallmann, 1946).

A close relationship between constitutional resistance to the schizophrenic genotype and graded resistance to pulmonary tuberculosis has been found. Only homozygous bearers of the specific (predisposing) genotype for schizophrenia are capable of reacting with a

TABLE 2

GENETIC FINDINGS IN PSYCHIATRIC CONDITIONS

Psychiatric condition	Investigator	General population	Half-sibs	Sibs	Two-egg twins	One-egg twins	Parents	Children	Postulated genetic mechanism
1. Manic-depressive psychosis	Kallmann	0.4%	16.7%	23%	26.3%	95.7%	23.4%		Autosomal irregular dominant
	Luxenburger			12.7%	1:16	31:33		24.4%	
	Rosanoff				11:67	16:23			
	Stenstedt			c. 15%			c. 15%	c. 15%	
2. Schizophrenia	Kallmann	0.85%	7.1%	14.2%	14.5%	86.2%	9.3%	16.4%	Autosomal recessive: 70% penetrance
	Slater				14.0%	76.0%			Recessive and dominant cases
3. Childhood schizophrenia	Kallmann and Roth			12.2%	17.1%	70.6%	12.5%		Similar to adult form
4. Involutional psychosis	Kallmann	1.0%	4.5%	6.0%	6.0%	60.9%	6.4%		Heterozygous carriers of schizophrenic genotype
5. Senile psychosis	Kallmann	Less than 1.0%		6.5%	8.0%	42.8%	3.4%		Gene-specific biochemical factors plus adaptive personality traits
6. Suicide	Kallmann *et al.*				1:28	1:28			None demonstrated
7. Adult male homosexuality	Kallmann *et al.*				42.3%	100%			Genetically disarranged balance between male and female maturational tendencies
8. Neurosis and psychopathic personality	Slater			M 2.4 F 0.0	M 42.1% F 7.4%	M 80.0% F 0.0%			Polygenic
	Conrad			4.0%	4.3%	86%			Polygenic (Kallmann)
9. Epilepsy	Alström			1.5%			1.5%	3.5%	Not genetic except rarely single dominant
	Gibbs *et al.* (EEG)				25%	100%			Single dominant
10. Mental defect (high-grade)	Brugger and Juda		23.4%	35.9%	54%	86%			Polygenic; special single recessive and single dominant forms
11. Mongolism	Lejeune, Gautier and Turpin								Trisomic condition demonstrated cytologically

true schizophrenic psychosis. Other features emerging from an analysis of the detailed figures of Kallmann's work are reduced reproductivity rate, increased rate of consanguinity in the parents and transmission in the collateral line of descent. Schizoid personality traits may be found in homozygotes with very high resistance or in heterozygotes with relatively low resistance (homozygotes having a double and heterozygotes a single dose of the gene).

Of the other serious workers and thinkers in this field none dispute the fact of heredity in schizophrenia and its great importance, but there are those who favor some other type or additional type of genetic mechanism. In Germany during the 1930's a simple dominant mode of inheritance was favored and became the basis for eugenic sterilization laws. Lenz, Koller, Slater and Böök still support the dominance theory as covering some or all of the cases.

Koller's argument cannot be presented here in detail, but it culminates in the contention that if the gene were recessive, the incidence of schizophrenia should be lower in the children than in the sibs, whereas the reverse is the case. Slater (1953*b*) likes this argument, inclines to the view that dominant as well as recessive forms exist, and adopts a view of genetic heterogeneity to explain the subgroups of schizophrenia, which Kallmann deals with more effectively, in my opinion, in terms of the modifying effect of the polygenic resistance mechanism. Penrose's theory of a multifactorial genetic determination of the disease does not conflict formally with Kallmann's hypothesis of a specific single recessive predisposing mechanism plus a multifactorial constitutional resistance mechanism, for after all one plus many equals many, and Kallmann's theory is much richer in detailed content and in its fruitfulness as a research hypothesis.

In bringing together findings in respect to manic-depressive psychosis and schizophrenia, it is interesting to note the cumulative effect of the evidence of the specificity and distinctness of the two genotypes. In reviewing the work of Kallmann's department in 1952, Hurst (1952) was able to report that among a total of 1,232 twin index cases no pair of one-egg twin partners was found whose clinical symptomatology warrants placing the two members of a pair into different diagnostic categories. Recently published observations of three different workers—Elsässer (1952), Slater (1953*a*) and Stenstedt (1952)—point in the same direction.

The relative infrequency of manic depressive psychosis in modern populations may be partly due to factors of selection which reduce the reproductive rate of the carriers without affecting the social level of their families (Kallmann, 1946; Stenstedt, 1952). At least there seems to be no tendency towards a social decline of the magnitude typical of schizophrenic family units.

3. *Childhood Schizophrenia.* In their paper entitled "Genetic Aspects of Pre-Adolescent Schizophrenia" Franz J. Kallmann and Bernard Roth (1956) provide the following conclusions from their study of 52 twins and 50 singletons under age 15 (quoted in part):

> (a) These findings indicate an early effect in childhood schizophrenia of the same genotype (gene-specific deficiency state) assumed to be responsible for the basic symptoms of adult schizophrenia. The conclusion is supported by the observation that the psychoses in the co-twins of early schizophrenia cases occur sometimes before and sometimes after adolescence.

(b) While the aetiological mechanism underlying the relatively infrequent activation of a schizophrenic psychosis before adolescence has not yet been adequately identified, it would seem to be connected with variable constellations of secondary factors lowering constitutional resistance.

(c) Because of a dearth of statistically comparable data, it is difficult to appraise the part played by a poor home with disturbed intra-family relationships in the aetiology of childhood schizophrenia as compared with that of adult schizophrenia.

4. *Involutional Psychosis.* According to Kallmann, the implication of comparative data "would seem to be that the principal genetic derivation of involutional psychosis is from an indirect relationship to the entity of schizophrenia, and not from a specific type of predisposition producing a particular impairment of the adjustive plasticity of aging persons."

5. *Senile Psychoses.* The severe maladjustment to aging resulting in the symptomatology of a senile psychosis would appear to depend on a combination of aetiological components, including age-susceptible personality traits, declining adaptational plasticity, and socioeconomic security. From their relation to those gene-specific biochemical phenomena which control growth and decline, these may be expected to coexist more frequently in genetically alike persons (one-egg twins) than in persons genetically considerably less alike (two-egg twins). The presensile brain atrophies of Pick and Alzheimer are probably both irregularly dominant.

The theme of maladjustment, examined from the point of view of the native assets or deficiencies which we bring to coping with our environment, is carried on in our next two topics of suicide and homosexuality. The former constitutes a total inability to adjust, and the latter, a socially inacceptable way of adjusting to one of our major instinctual drives.

6. *Suicide.* In suicide, that final and irrevocable failure to adjust to life, there has been a widespread tendency to assume an inheritable type of unfitness in the general personality structure. This has not been borne out by the investigations of Kallmann and his co-workers (Kallmann and Anastasio, 1946; Kallmann *et al.*, 1949). The first 27 sets of twin subjects were discordant as to suicide. In 1950 this run was broken by a pair of schizophrenic and overtly homosexual World War II veterans, who became concordant as to suicide, albeit at different ages—25 and 29—and by different methods—drowning and gas. A single instance of this type in a sample of this size is clearly within the range of chance expectation. The general conclusion from this study of suicide in twins, as well as from another one by the same group in only children, is that there is no statistically valid support for the notion of hereditary or familial occurrence of suicide.

7. *Adult Male Homosexuality.* The twin family method provides a means of contributing to a scientifically validated elucidation of this condition, in which highly speculative psychodynamic theories have long held the field. Kallmann (1952) in his study used Kinsey's 6-point scale in rating adult male homosexuality.

One salient finding (reflected in Table 2) was that in 40 one-egg twin pairs showing a high degree of homosexuality (Kinsey ratings of 3 or more) there was 100% concordance for the trait in question, while for the 45 two-egg pairs the concordance is 11.5% or 42.3%, according to whether the two highest homo-

sexual ratings (5 and 6) or the whole range (1-6) are taken as the criterion. (These figures are only slightly higher than Kinsey's estimates of 10% and 37% of the total population.) A point of further interest is the fact that most of these twin pairs claimed to have developed their sexual deviation independently and far apart from each other, and that all of them denied categorically any history of mutuality in overt sex relations.

An important conclusion also emerges from a consideration of sex ratios in the sibships of homosexuals (not reflected in Table 2). In the single-born and twin-index samples alike, there is a fairly consistent tendency for this to deviate towards an excess of males. In the sibships studied by Kallmann a ratio of 126 males to 100 females was obtained (as compared with 106 to 100 in the corresponding general population). This is in line with Lang's demonstration of a statistically significant deviation in the direction of a preponderance of males in the sibships of a large sample of some 1,015 German homosexuals, where the ratio for all ages was 121.1 males to 100 females and, for subjects over the age of 25, 128.3 males to 100 females. The inference from this is that adult male homosexuality is a biologically determined phenomenon on a genetic basis—due to a genetically disarranged balance between male and female maturational tendencies.

8. *Psychoneurotic Traits, Psychosomatic Vulnerabilities, and Psychopathic Personality Traits.* As to possible genetic elements at the root of psychoneurotic traits and psychosomatic vulnerabilities, it is regrettable that current information in this important area of psychiatric genetics is least complete. In Eysenck's (1947) opinion many differently formulated theories of neurosis are on the same descriptive level and are concerned with the same fundamental dimension of personality. In collaboration with Prell (Eysenck and Prell, 1951), he was one of the few investigators of psychoneurotic reaction patterns (outside the field of criminality) to avail himself of the opportunities afforded by the twin-study method. On the basis of their study of 25 one-egg and 25 two-egg pairs, Eysenck and Prell classified "the neurotic personality factor" as a biological and largely gene-specific entity, estimating the genetic contribution to this neurotic unit predisposition at 80%.

Slater brings together neurotic and psychopathic reactions, viewing them clinically as different grades of maladjustment to stress. Genetically he considers that his figures suggest the hypothesis of nonspecific (polygenic) deficiencies in relation to stress. In the psychoneuroses he found that the form of the symptoms is not as closely related to the form of stress as it is to the basic personality. EEG findings (theta activity) suggest a heredito-constitutional basis for psychopathic personality, especially the aggressive variety, and raise the possibility that it is literally a form of physiological immaturity of the central nervous system. Slater moreover cites a study in which there is a much higher rate of psychopathic personality in the offspring of psychopaths than in the general population.

9. *Epilepsy.* In the field of epilepsy the evidence from various sources is conflicting. The presumption is that the tendency to seizures is a graded character (Kallmann and Sander, 1947)—varying from the potentiality of all the human species (and indeed many subhuman organisms) to develop seizures under the influence of unusually potent

stimuli such as that employed in electroconvulsive therapy, through the cases where rather special irritant agencies such as alcohol, fever or trauma are sufficient to elicit seizures, to the clinically recognized epileptics who have their seizures under the range of stimuli provided by the everyday environment. This would suggest the likelihood of a polygenic mechanism for any hereditary basis involved. The figures in Table 2 (Conrad, 1935) provide substantial evidence for a genetic basis and are not incompatible with the polygenic hypothesis which is favored by Kallmann. The figures of E. L. and F. A. Gibbs (Lennox *et al.*, 1940, 1945), based not on a clinical but on an EEG criterion — dysrhythmia—are interpreted by them quite unexceptionably as favoring single dominance. The question is further beclouded by Alström's (1900) finding of an overall incidence figure for children, parents, and siblings of the same order as that characteristic of the general population concerned. These results would suggest either that in general no genetic factor is discernible or that we have to do with a major dominant gene, of low penetrance owing to powerful modifiers. Detailed scrutiny of the results reveals that in only about 1% of his 897 index cases—11 probands belonging to 8 families—does the epilepsy appear to follow a single dominant course with high penetrance.

10. *Mental Deficiency.* Mental deficiency is clearly not a homogeneous disease entity but the end result of a variety of possible factors or entities, genetic and nongenetic. Penrose (1949), in his Colchester study, showed that at least 15% of cases were nongenetic inasmuch as they had resulted from environmental causes such as birth injury or encephalitis.

Of the cases with a substantial genetic basis, the large majority (more particularly of the higher grades over IQ 50) may be regarded as the tail-end of the curve of normal distribution of intelligence, based on a polygenic mechanism. It is not suggested that intelligence is entirely dependent on genetic factors, but rather that insofar as a genetic mechanism is involved it is polygenic. The twin studies of Newman *et al.* (1937) indicate that genetic constitution contributes twice as much as the environmental factor of cultural stimulation to test intelligence. In lower-grade mental deficiency (IQ of less than 50) special types of mental defect based on single recessive or single dominant mechanisms feature prominently. Examples of single recessive mechanisms are amaurotic idiocy and phenylpyruvic oligophrenia, and of single dominant mechanisms, tuberous sclerosis, neurofibromatosis and Sturge-Weber's disease.

According to Brugger and Juda even certain types of high-grade defect are single-recessive as an explanation of the morbidity figures appearing in Table 2.

That pedigree studies can easily be confused by selective factors is nowhere better demonstrated than in the field of mental defect—the classical sagas of the the Jukes and Kallikaks are no exception to this pronouncement.

11. *Mongolism.* This condition is worthy of separate mention both because of the very special clinical interest attaching to this class of case, and because it was here that cytological genetics achieved a famous victory in the demonstration by Lejeune, Gautier and Turpin (1959) of a small extra chromosome in tissue cultures from the connective tissue of three cases of mongolism. This was confirmed a little later during the same year in respect to the somatic chro-

mosomes by Jacobs *et al.* (1959) in six typical mongols, three of each sex.

12. *Huntington's Chorea.* The claim of this condition to transmission as a Mendelian dominant is so well known that it will not be elaborated here. The trait is specific, autosomal, and single-dominant.

A General Perspective on Psychiatric Genetics

The position historically as I see it is as follows:

In the last century crude hereditarian views were obtained in the field of mental disorders, in which a nonspecific type of heredity, related to the so-called neuropathic diathesis, was supposed to be operative—and which could, it was alleged, bring about in some inexplicable fashion the appearance of mental disorder A (say schizophrenia) in the first generation, mental disorder B (say manic-depressive psychosis) in the second generation, and mental disorder C (say epileptic psychosis) in the next.

During the present century there was a reaction away from such a crude view which flew in the face of plain logic. The very development of scientific genetics during this period disclosed the untenability of such a view.

Then there was the environmental bias of special influential schools of thought, notably psychoanalysis and behaviorism. This created an atmosphere inhospitable to genetic ideas which, as then conceived, denoted irreversibility. There was a general human rebellion against the notion of determinism in human behavior, especially of a determinism that stretches from generation to generation.

With the advent, however, of the more precise studies that we have reviewed, and the new concept of specificity of genetic mechanisms for different diseases with this dynamic biochemical basis and consequent reversibility, the currency of genetic ideas in psychiatry has steadily been gaining ground.

Perhaps we can give the best expression to the most significant emphases in the orientation of modern psychiatric genetics by quoting excerpts from a paper by Kallmann (1953*b*).

> Along with the advent of modern psychiatry at the end of the last century, it became necessary to look for genetic components in the aetiology of psychotic behaviour patterns. . . . . By its very nature, the genetic approach ended those carefree days when mental illness, for want of a better label, was called lunacy and ascribed to the disturbing effect of the idle goddess.
>
> Generally speaking, psychotic behavior disorders seem to follow selective patterns of distribution and cannot be explained either by a universal species vulnerability or by general emotional group experiences. Since these disorders affect only some members of some families, under nearly equal conditions of stress, it follows that the key to persisting obscurities in their aetiology must be sought in certain specific disarrangements of those maturational integrative or regulative functions which determine the adaptive capacities of the human organism.
>
> In conclusion, it may be said that in order to understand certain basic principles in the formation of pyschotic behaviour patterns, it is necessary to delve into the substratum of gene action as well as into the subconscious.
>
> . . . . . In doing so, genetic studies have in many instances succeeded in conferring the belief, at least theoretically, that mental disorders are both preventable and potentially curable. At the same time, such studies have again focused attention on the importance of a systematic and well balanced approach in any attempt to reach the given goals. It is now clearer than ever that only when attitudes of optimistic complacency toward the

causes of severe maladjustment give way to a realistic awareness of the incompleteness of knowledge regarding the genetic aspects of psychotic behaviour patterns, will further progress be made.

Genetic Findings in Neurological and Other Disorders

1. *Neurological.* The subjoined list of neurological (and allied) disorders gives some indication of the wide applicability of the genetic theme in this field.

(a) Usually single-dominant syndromes: Marie's heredocerebellar ataxia; status dysrhaphicus (syringomyelia); hereditary essential tremor; congenital cataract, glaucoma and bilateral ptosis; paralysis agitans; Thomsen's myotonia congenita; familial periodic paralysis and some forms of muscular atrophy (Charcot-Marie).

(b) Usually single-recessive syndromes; Friedreich's ataxia (spinal form); Wilson-Wesfal's pseudosclerosis (progressive lenticular degeneration); hereditary congenital deafness; most forms of muscular atrophy (Djerine-Motta) and muscular dystrophy Duchenne-Erb, and spastic spinal paralysis.

(c) Usually sex-linked syndromes; Leber's primary optic atrophy; retinitis pigmentosa; haemophilia; familial eunuchoidism associated with anosmia, and color blindness.

2. *Other Disorders Due to Genetically Determined Defects*

(a) Storage diseases (single-recessive); Gaucher's lipoidosis; Niemann-Pick's acute xanthomatosis; Hand-Schüller-Christian's chronic xanthomatosis; Gierke's glycogen disease; congenital porphyria.

(b) Rh blood incompatibilities causing erythroblastosis and possibly certain undifferentiated forms of mental defect.

A General Perspective on Neurological Genetics

Neurology is richly illumined by the insights provided by genetics. Not only is the basic aetiology of the degenerative and some other neurological conditions laid bare, but clinical insights relating to varying penetrance and expressivity and the disentangling of features of mixed syndromes are made possible. As in psychiatry, the adding of biochemical dimension to genetics bids fair to open vistas to research and therapy in conditions hitherto regarded as incurable. We have had glimpses of this biochemical aspect in the foregoing, but we shall conclude this section with one of the clearest examples of a known enzyme block in a condition which happily falls into the sphere of both neurology and psychiatry, namely phenylketonuria or phenylpyruvic oligophrenia.

George A. Jervis (1953) gives the following summing up of the biochemical nature of phenylketonuria and the relationship, if any, of the metabolic error to the clinical symptomatology.

The Biochemical Nature of Phenylketonuria

Several hypotheses have been advanced.

1. The error consists of a block in the metabolism of phenylalanine at the stage of phenylpyruvic acid; . . . . .
2. The error consists of an abnormal racemization of phenylalanine. . . . . .
3. The error consists of a blockage in the conversion of phenylalanine into tyrosine, the unmetabolized amino acid would then accumulate in the body fluid and be excreted as such or as its deaminated products, phenylpyruvic, phenyllactic and phenylacetic acid.

Jervis cites the experimental findings

which make him support the last hypothesis.

He goes on to analyze the question of the relationship, if any, of the metabolic error to the clinical symptomatology and in conclusion stresses the distinct possibility that the relationship is not a casual one, and that the two phenomena, biochemical and mental defect, may be the expression of a genic action pleiotropic in nature.

With a view to more precise pinpointing of biochemical pathways and their potential interrelationships with the mental deficiency present, Pare, Sandler and Stacey (1959) have conducted an investigation with restricted phenylalanine diet (generally beneficial for this condition) on 7 in-patients at a children's hospital. Serum and urinary 5-hydroxytryptamine were observed to rise in all but one case. They have also conducted a 5-hydroxytryptophan tolerance test, in which 4 phenylketonuric children matched with 4 children with simple mental defect, that gave results in accord with the in vitro findings of Davison and Sandler (1958) that certain aromatic acid metabolites of phenylalanine found in excess in phenylketonuria will inhibit 5 HTP decarboxylase: restricted phenylalanine diet by decreasing the production of these metabolites would be expected to correct such an inhibition. Of their failure in a six-month controlled trial of 5-hydroxytrytophan administration to 8 young phenylketonurics to raise the I.Q.'s, Pare *et al.* remark that "it must be remembered that other enzyme systems may be similarly inhibited by the abnormal metabolites so that a similar decrease in catechol production, for example, may play a part in the pathogenesis of the mental defect."

Implications for Research and Therapy of Model Psychoses and Neuropsychopharmacology Interpreted in the Light of Enzyme Blocks

We shall now refer to the work on model psychoses, which have become so popular of late, with a view to exploring possible interrelation with biochemical genetics and to tracing the significant enzyme blocks of possible aetiological significance in neuropsychiatric conditions.

In a previous communication, Experiences with D-Lysergic Acid Diethylamide (Hurst *et al.*, 1956) we have raised certain methodological issues that arise in connection with such work, which we shall now set forth:

1. A precise characterization of the picture presented in the course of drug-induced psychoses is essential. The history of the earlier work shows that the psychoses induced by LSD and allied agents were uncritically and schematically identified with schizophrenia, whereas careful evaluation of our own cases, as well as the literature, shows that manic-depressive and toxic psychotic reaction components also occurred, either as the predominant feature of an individual case or as only a phase within the reaction of one individual.

2. These experiments are likely to be most rewarding in the light of full studies of the personalities concerned, which become prepsychotic personalities in relation to drug psychoses. A global description of these personalities in terms of the cyclothyme/cycloid and schizothyme/schizoid scheme is important, as well as in more detailed terms of the independent variables of temperament and personality and in psychodynamic terms.

3. The more global assessments provide a basis for determining whether schizophrenic-like or manic-depressive-like features of the psychosis constitute primarily a drug effect or merely the release of underlying personality tendencies. In the one case a more spe-

cific role for the chemical concerned for one of the psychoses would be implied, while in the other a common chemical involved in aetiology and manifesting differently in persons of differing psychological and physical constitutions could be inferred.

4. Careful comparisons between the reactions to various drugs such as LSD, mescaline and adrenochrome would be of value in narrowing down the biochemical quest for the hypothetical enzyme block or blocks involved in the model or naturally occurring psychoses.

Growth of neuropsychopharmacology has been phenomenal since the last-named article was written. Of central significance to the elucidation of the alternative formulated in Paragraph 3 above has been the demonstration of the selective action of the mono-amine-oxidase inhibitors and imipramine on the depressive phase of manic-depressive psychosis, vis-a-vis schizophrenia. I have developed this issue more fully in a paper read before the Society of Medical Sciences of Lisbon in November 1959 (Hurst, 1959).

Biochemical work within the sphere of schizophrenia itself, although unsynthesized, is nevertheless substantial. This work, in principle, is in line with the general genetic-biochemical-enzyme aetiological hypothesis that is emerging from the other disciplines of psychiatric genetics and neuropsychopharmacology which we have been considering. It is for research of the future, and it would appear the near future, to discover and forge the significant links between the contributions from these three spheres. Within biochemistry itself, Seymour S. Kety (1959) has done us the service of disentangling central strands in this work in a critical review of biochemical theories of schizophrenia and of the evidence to support them, examining in turn (a) Oxygen, Carbohydrate Metabolism and Energetics, (b) Amino Acids and Amines, (c) The Epinephrine Hypothesis and (d) Taraxein and its forerunner Ceruloplasmin. Consideration of details is beyond the scope of the present article, but one may be permitted the observation that the demonstration by Leach and Heath (Heath *et al.*, 1957) and their co-workers of the induction in normal volunteers of transient schizophrenic states, carefully described in terms of Bleuler's primary and secondary symptom categories, by the injection of taraxein-containing schizophrenic blood serum is a finding with a claim to rank among the major dramatic episodes in the history of medical discovery. Wada and Gibson's (1959) work on behavioral changes in cats and monkeys resulting from schizophrenic urine extracts offers an interesting parallel to the human taraxein work.

New developments in the basic chemistry of chromosomes and genes may at any moment afford coordinating and detailed insights into the field of biochemical genetics. Of particular interest in this connection are current advances in our knowledge of the structure of nucleic acids in relation to gene action, and the Watson-Crick model of DNA (deoxyribose Nucleic Acid) illustrating the pairing of purines and pyramidines along two complementary strands. There are also important implications for Man in microbial genetics and "viral" genes, because the chemical basis of the genes is essentially similar in all living entities down to the simplest viruses.

Zamenhof (1959), in analyzing the concept of enzyme blocks which occupies so central a position in explaining hereditary defects in Man, points out that failure or lack of an enzyme does not necessarily imply that the molecule of the enzyme (or of the protein in gen-

eral) is missing altogether: it may mean that it is defective, i.e., changed so as to be partially or totally inactive. He goes on to show that all the change necessary in the protein underlying a mutation may be the substitution of one amino acid out of hundreds. Thus the haemoglobin modification resulting in the production of sickle cell anaemia involves the change of only one amino acid unit out of 300—glutamic acid is replaced by valine. This, he stresses, is all the mutation had to do to make itself felt, and he is tempted to the speculation that the corresponding change in the hereditary determinant, the DNA molecule, was just as small — perhaps a change in a single nucleotide.

THE ROLE OF EUGENICS AND HEREDITY CLINICS IN RELATION TO NEUROPSYCHIATRIC CONDITIONS

Although we find eugenics in Plato's *Republic* and practical experiments in this direction in Sparta and in the Oneida Community in Vermont dating from 1841, the first exponent of systematic eugenics in modern times was that eclectic genuis Francis Galton (1869, 1874, 1883, 1889, 1908). Inspired by the evolutionary vista provided by the publication in 1859 of the "Origin of Species" by his cousin Charles Darwin, and the striking cases of apparently hereditary genius among Cambridge men who had been at the University at about his time, he first evolved the statistical theory and established the anthropological laboratories necessary to the study of man as a science. Then, feeling very strongly the importance of applying his findings to race improvement, he launched out into the dissemination of eugenic ideas.

It is clear from his writings that Galton's program included both negative eugenics — the elimination of stocks afflicted with serious hereditary disease— and positive eugenics—the favoring of propagation of stocks of superior abilities by providing suitable inducements to this end.

The subjoined formulation of his advocacy of negative eugenics shows his concern for doing so in a democratic setting (Galton, 1908).

> As in most other cases of novel views, the wrongheadedness of objectors to Eugenics has been curious. The most common misrepresentations now are that its methods must be altogether those of compulsory unions, as in breeding animals. It is not so. I think that stern compulsion ought to be exerted to prevent the free propagation of the stock of those who are seriously afflicted by lunacy, feeble-mindedness, habitual criminality and pauperism, but that is quite different from compulsory marriage. A democracy cannot endure unless it be composed of able citizens; therefore it must in self-defence withstand the free introduction of degenerate stock.

Relative to positive eugenics he says (Galton, 1908):

> I also hope that social recognition of an appropriate kind will be given to healthy, capable and large families, and that social influence will be exerted towards the encouragement of eugenic marriages.

Galton (1908) repeatedly stresses the importance of eugenic theory and practice. Thus he says:

> I hence conclude that the improvement of the breed of mankind is no insuperable difficulty. If everybody were to agree on the improvement of the race of man being a matter of the very utmost importance and if the theory of hereditary transmission of qualities in men was as thoroughly understood as it is in the case of our domestic animals, I see no absurdity in supposing that in some way or other, the improvement would be carried into effect.

And again (Galton, 1908):

> I take Eugenics very seriously, feeling that its principles ought to become one of the dominant motives in a civilised nation.

Galton's advocacy of eugenics never degenerated into fanaticism or bigotry, as can be inferred from such statements as the following (Galton, 1908):

> What I desire is that the importance of eugenic marriages should be reckoned at its just value, neither too high nor too low, and that Eugenics should form one of the many considerations by which marriages are promoted or hindered, as they are by social position, adequate fortune, and similarity of creed;

In his "Enquiries into Human Faculty and Its Development" (Galton, 1883) published in 1883, he emphasizes that:

> The eugenic aim is the amelioration of average human qualities through the observation of heredity, rather than the production of a few exceptional record-breakers.

It is clear that when we are considering a program of negative eugenics for the elimination of serious hereditary disease (in our case serious hereditary mental disorder or defect or incapacitating neurological disorder), the new vista provided by physiological and biochemical genetics immediately raises an approach which limits or negates negative eugenics—for if genetics points the way to curing a condition by locating the significant enzyme blocks and reversing them, are we justified, or indeed is it logical, to attempt elimination by eugenic means? The answer is unequivocally in the negative as I see the matter. We can therefore regard negative eugenics merely as a temporary measure pending the fruition of our more fundamental hope.

With this limitation let us consider some features of a program of negative eugenics, with special reference to neuropsychiatry.

1. In the first place action is clearly only indicated where the trait is very seriously disabling and incurable.

2. Second, negative eugenic action must be based in each individual case on the specific genetic mechanism reflected in the empiric risk figures. In a single dominant condition such as manic-depressive psychosis we are clearly confronted with a different situation from that in schizophrenia, where a single recessive mechanism is postulated. In the first case we have transmission of the disease in the direct line of descent, manifestation in approximately half of the offspring, the other half not carrying the gene and being incapable of transmitting the disease to their offspring in turn ("once free forever free"). In the second case, however, most schizophrenics are the children of overtly normal parents, each of whom are carriers of one schizophrenic gene. The manifestation rate in such children is 16% (the 25% expected for full penetrance being reduced by the 70% penetrance of the schizophrenic genotype). Overtly unaffected children, as well as brothers and sisters of the first generation, are a rich source of carriers of "one gene," ready to manifest in the case of marriage with a similarly constituted person. This fact, coupled with the finding that 70% of the children due to be born, on the average, to a schizophrenic are born to him before the disease manifests and he is hospitalized for it, accounts for the low estimate that only 2% to 4% of schizophrenics would be eliminated by insuring that the affected schizophrenic does not reproduce. This figure may well be increased when the mapping of the human chromosome now in progress becomes more complete, enabling us to identify the potential schizophrenic before the condition develops. If, however, we wish to follow the logic of the situation quite ruthlessly, we are compelled to concentrate on the brothers, sisters and children of schizo-

phrenics as the crux of the eugenic situation.

Cousin marriage, or marriage into other schizophrenic families, or marriage with a markedly introverted individual, is especially undesirable for members of schizophrenic families, as being likely to bring two recessive genes together to produce a proportion of frank schizophrenics.

Where the type of hereditary mechanism is in dispute, the empiric-risk figures form an objective basis for eugenic procedure.

3. Third, as regards the procedure of attaining the non-propagation of the scientifically contra-indicated types of person, let us lay the ghost of sterilization at the very outset. Sterilization is so outmoded in the sphere of eugenics that at the 1956 Congress at Copenhagen there was not one reference to it! No, the modern approach in our democratic societies is to advise the afflicted person and/or his spouse (the latter particularly in the case of mental illness where the patient himself may not be capable of understanding the position) what the risk is, in percentage terms, that any contemplated child or children will be affected by the hereditary trait concerned. Kallmann's experience with heritable mental disease is that in the metropolitan communities in which he has worked the application of birth control methods has been quite adequate to the objective envisaged, rendering the consideration of sterilization unnecessary. In cases where religious objections render contraceptive methods unacceptable, abstention from marriage is often recognised as the best course to pursue.

HEREDITY COUNSELING

This brings us to the question of heredity counseling services and centres. Helen G. Hammons (1956) lists 15 such centres for the United States and Canada, 1 in Denmark, 5 in England, 1 in France, 1 in Germany, 2 in Holland, 1 in India, 2 in Italy, 1 in Japan, 1 in Norway, 3 in Sweden, and 1 in Switzerland; two of these are primarily concerned with neuropsychiatric cases.

Dr. Lee R. Dice (1952) in a presidential address of the American Society of Human Genetics gave much data about the functions and operations of such clinics, which I now proceed to summarize:

(a) *The problems presented to clinics* are diverse, including questions from engaged couples with possibly hereditary disease in one or both families, married couples with one or more children already affected by possibly hereditary disease; and children's placement bureaux regarding chances of the child showing an adverse parental trait.

(b) *Drawing up of kindred pedigrees* includes a system of record sheets with data by sibships, graphic pedigrees and sources of information.

(c) *Mode of inheritance* is then postulated on the basis of genetic principles.

(d) *First-hand examination of near relatives*, by a competent specialist if necessary. This involves financial considerations.

(e) *Records and documents* should be detailed and precise to be understandable 10 or 100 years hence. Abbreviations and scientific jargon are to be avoided. Professional persons referring the case should be encouraged to supply detailed written reports, including a formulation of the problem.

(f) *Professional secrecy and confidence.* Special care should be taken in this regard concerning the disclosure, even to relatives, of illegitimate births, adoptions, age of elderly women, and the results of clinical examinations not duly authorised by parent or guardian.

(g) *Information and advice to patients (clients).* This is limited to the restrained technique of stating the risk (in percentage terms) of the trait under consideration. Under no circumstances should the counsellor tell a couple whether they should or should not have a child. In certain cases he will have to confess that he can make no sort of prediction on the basis of the information available. In many cases he will be in the happier position of being able to reassure the parents and advise them that there is no

risk of the feared hereditary disease manifesting in the children.

(h) *Weight given to positive eugenics and desirable hereditary traits.* Although the heredity clinic focuses its attention on the elimination of undesirable traits, the counsellor does not lose sight of positive eugenics, e.g., in weighing up positive qualities of intelligence and temperament against an only moderately undesirable genetic trait.

(i) *Duty of publication.* Inasmuch as each heredity clinic acts as an archives of hereditary disease, there is a duty of publication so that human geneticists the world over may pool their information, especially as regards rare traits.

(j) *Duty of education.* Comparatively few medical schools instruct their students in genetics, especially human genetics, and yet, with the conquering of infectious and nutritional disease, heredito-constitutional disease is growing in volume and relative importance. Every heredity clinic should therefore provide lectures for undergraduates and provide facilities to direct the work of postgraduate students.

(k) *Organization of clinic–practical aspects.* The minimal staffing is one geneticist, one physician, one laboratory assistant and a secretary. There should be access to various clinical laboratories and a liaison with clinics of a well-equipped hospital. The minimum space is a general office, a waiting room and one office for each member of staff. It is, however, possible to share with other units a medical examination room, laboratories, photographic studio and dark room, library and seminar rooms. Fees should not be asked from patients or clients, except that fees for cooperating specialists should be paid when they can be afforded. The budgeting includes provision for travelling by distant relatives coming for examination and an item for staff transport for field work, as well as for staff salaries.

Sheldon Reed's (1955) book on hereditary counseling and that edited by Helen G. Hammons (1959) are commended to those requiring a more detailed picture of heredity clinics.

POSITIVE EUGENICS

Little more need be said about positive eugenics. Eugenicists have been alarmed by the apparent trend of persons of lower ability to have on the whole larger families than those of higher ability. Positive eugenics advocates attempting to reverse this trend by devising ways of encouraging persons of above-average ability to marry earlier and to have larger families. It is recognized that such an objective cannot be tackled in an isolated fashion, but takes us deep into problems affecting the structure of society as a whole. Kallmann summarizes positive eugenic measures as follows:

(a) Securing larger families from eugenically desirable classes:
   1. by educational methods
   2. by economic support of eugenically desirable families (proper houses at low rentals), and
   3. by increasing the income of eugenically desirable families at the birth of each child, by differential taxation in relation to family size, and by lowering the wages of single and childless workers.

(b) Birth control measures as practiced for sociological reasons.

Viewing the progress of eugenics historically there have been signs in recent years of sound and vigorous development of a separate science of human genetics, embodied in such concrete ways as the foundation in 1949 of the American Society of Human Genetics, with its own journal, following on the pioneer work of the journal *Eugenical News* (now *Eugenics Quarterly*) under the leadership of Frederick Osborn, the formation of the Population Council, Inc., the institution of the heredity clinics

mentioned above, as well as academic centres like the Galton Laboratory (University of London), and the holding of the First International Congress of Human Genetics at Copenhagen in 1956.

Frederick Osborn (1956), in an article entitled "Galton and Midcentury Eugenics," gives point and perspective to the advance in eugenics that we are considering by attempting to appraise how the founder of modern eugenics would react to the situation if he were to return to the present-day scene:

> Galton, who died in 1911, would have been fascinated with the last half-century's progress in the science of genetics, which had become a subject of controversy during the last few years of his life. He would have given his powerful backing to the further advancement of human genetics. He would have been present at the . . . . . Congress to be held in Copenhagen . . . . He would have urged exhaustive studies on the identification of carriers of harmful recessive genes. He would have supported heredity clinics.

While Galton would have been disappointed at the little progress that has been made in the study of the genetic factors which affect the development of personality and intelligence, Osborn feels he would have been pleased with the methodologies of modern psychology, of which he is one of the pioneers, and with the applications of new measures in personnel selection. He would have been puzzled by the relatively small pure-research appropriations for the social as compared with the physical sciences. Osborn, in elaborating Galton's emphasis on directing "forces under social control," feels that he would not have been skeptical of the possibility of establishing the differential of larger families for the better endowed. To achieve his aims Osborn feels that eugenics will have to drop the idea of assigning genetic superiorities to social or racial groups, and even designating individuals as superior or inferior. "Let's stop telling anyone that they have a generally inferior genetic quality, for they will never agree. Let's base our proposals on the desirability of having children born in homes where they will get affectionate and responsible care, and perhaps our proposals will be accepted." This attitude clearly squares with Galton's emphasis that eugenic policies would fail unless geared to "existing conditions of law and sentiment."

Osborn concludes: "It seems to me that if it is to progress as it should, eugenics must follow new policies and state its case anew, and that from this rebirth we may, even in our own lifetime, see it moving at last towards the high goals Galton set for it."

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SOME DATA ON NATURAL FERTILITY

LOUIS HENRY

WE CAN term as natural the fertility which exists or has existed in the absence of deliberate birth control. The adjective "natural" is admittedly not ideal but we prefer it to "physiological" since the factors affecting natural fertility are not solely physiological: social factors may also play a part—sexual taboos, for example, during lactation. Some of these factors may result in a reduction of fertility but this cannot be considered a form of birth control. Control can be said to exist when the behavior of the couple is bound to the number of children already born and is modified when this number reaches the maximum which the couple does not want to exceed; it is not the case for a taboo concerning lactation, which is independent of the number of children already born.

It is no easy task to collect data on natural fertility, thus defined. One is confronted in fact with two difficulties.

First of all, populations have to be found where birth control is not practiced, or populations where control is so limited that it does not effectively modify fertility. The latter condition would appear to be less restrictive but actually it is very difficult to be certain that a given population fulfills these requirements. In the great majority of cases, one simply forms a hypothesis based on considerations of time, place, thought, culture, type of society, etc. Thus one comes to adjudge as sufficiently approximating to natural fertility the fertility found in the rural population of Europe or in peoples of European origin before a certain date, which itself varies from one country to another and, within one country, from one region to another. In the case of France, the date is usually fixed between 1750 and 1790; for other countries, 1880 is about the earliest date that can be cited. Urban populations are more difficult to place, but most difficult of all are the governing classes about whom it seems to be impossible to make an evaluation without a preliminary study of the development of their fertility. Such a study, carried out on the governing classes of Geneva, indicated that fertility was close, even very close, to natural fertility in the generations born before 1650, but a study of the French aristocracy living at Court did not reveal a similar situation; perhaps it would be found if it were possible to go further back in time.

For non-European populations there is often strong evidence to suggest virtually complete absence of birth control, either for the immediate past or up to the present. This evidence is further reinforced by studies showing that the very idea of limiting births is more or less foreign to them.

The second difficulty lies in the collection of data, for, excluding the Hutterites of the United States and Canada, the populations which presumably did not or do not pratice birth control were either premodern or inhabit countries where demographic statistics are not developed and are of poor quality. Spe-

cial efforts are needed to collect relevant data on the fertility of these populations, and they have only been made in recent years; just ten years ago very few data were available. Since then, however, the situation has considerably improved thanks to the work of historical demographers on the one hand, and fairly numerous studies of the underdeveloped countries on the other.

The term "fertility" can be interpreted in several ways: it can be used to designate a demographic characteristic which is affected by marriage rates, and legitimate and illegitimate birth rates. This composite, or general, fertility is not well suited for analytical, and is even less suited for comparative, purposes because of the variety of situations possible within the state of marriage.

A more valid comparison can be made by restricting the study to legitimate birth rates, or the equivalent; that is, the fertility of women who have formed a union which is stable enough for them to be considered as exposed to the same risk as married women. Moreover, the age of the woman has to be taken into account as that also plays an important part. In other words, rates for legitimate births by age or age group must be formulated.

In populations where birth control is widely practiced these rates are of real interest only if women are classified by age or age group at marriage; the number of children born at the time of the study, and therefore the duration of the marriage—or the difference between the age at the time of the study and the age at marriage — then have an important bearing on the case. But it is a different matter where birth control is absent or little practiced. In Taiwan, India and in the majority of populations of traditional, rural societies for which it has been possible to deduce the age at marriage, it has been established, by studying a group of women of the same age, that fertility was more or less independent of the age at marriage.[1, 2]

One can proceed, therefore, to the stage of making comparisons without introducing the age at marriage; it is in fact an advantage since in the underdeveloped countries the age is rarely known. The 15-19 age group must be excluded, however. This group is always close to marriage (see footnote 2); on the other hand, and this is the more important point, fertility increases rapidly with age before 18 or 20, with the result that the fertility of the 20-24 age group depends on the average age at marriage of those women married before 20; and this age may vary considerably from one population to another.[3]

Since fertility measured by age group does not depend on the age at marriage, at least after the age of 20 or thereabouts, the sum of the age-specific fertility rates multiplied by 5 equals the mean number of children, discounting mortality, per woman married at 20.[4, 5] This mean number constitutes a sort of guide-mark for the legitimate fertility level of the

[1]Canada forms an exception (marriages from 1700-1729), but certain anomalies lead us to suppose that the connection between fertility and the age at marriage is only apparent.

[2]Theoretically this is true only after a sufficient number of years of marriage have passed for the fluctuations in fertility at the beginning of the marriage to have been levelled out. Calculating by age group renders this condition unimportant.

[3]No difficulties are resolved by working from the exact age, for the reservations expressed in footnote 2 then demonstrate their full force.

[4]The multiple 5 is used because the rates by age group represent the average for 5-year periods.

[5]This average number differs very little from what can be observed in the completed families of women married at 20 years, because the connection between fertility and mortality is in fact slight.

age groups from 20 to 50 years. As such it can be used to classify populations according to their fertility.

This is what has been done in Table 1, which gives for different populations a series of legitimate fertility rates from the 20-24 age group to the 45-49 age group, and the average number of children per woman married at 20 corresponding to each series. The populations considered are listed in descending order of the average number of children. In the right-hand column, the indices are listed: the arithmetic mean of the rates for the two groups 30-34 and 35-39, and likewise of the two groups 40-44 and 45-49, has been computed and compared with the arithmetic mean, taken as base 100, of the 20-24 and 25-29 groups. This has been done for each population.

The index of mean rates for women in the 30-39 age group ranges from 67 (Fouta-Djalon) to 89 (Canada) but of a total of 13 series, 9 fall within the range 76 to 81.

The index of mean rates for women in the 40-49 age group ranges from 14.5 (Sotteville-lès-Rouen) to 28.5 (Norway) and there are only 6 series within the 17-22 range; the relative dispersion is far more marked than in the former group. But the difference is partly apparent and can be attributed to errors in recording age and to random fluctuations; both factors carry more weight for the 40-49 age group than for the 30-39 group.[6]

[6]Difficulty in determining age is most often encountered in underdeveloped countries. The age group defined as 45-49 always includes women who are in fact younger and consequently a fertility rate which appears to be too high is obtained. The inclusion of women older than their stated age has only a slight compensatory effect since fertility in the 45-49 age group is almost nonexistent.

Among traditional societies, either European or of European origin, the age can be precisely reckoned, or calculated from the age at death. In this latter case, one runs the same risk as above.

Despite the dispersion, there does exist some similarity between the different series: they have only to be compared with the series of rates for a population where birth control is widely practiced (last line of Table 1) for this to be obvious. Therefore they can all be included in a general description when examining the average of the rates; a series that is more evenly graded than any of the component series is thus obtained. Taking the fertility of the 20-24 age group as base 100, the rates are as follows: 25-29 years, 93.5; 30-34 years, 85.3; 35-39 years, 68.7; 40-44 years, 34.9; and 45-49 years, 5.

Table 2 compares this average series of indices with that of a population where birth control is widely practiced. The first line remains fairly steady up to about 35 years and then drops sharply. The second line shows a typical decline—from one age group to the next, the fertility rate is almost halved.

Sterility. All or nearly all couples are sterile by the time the woman is 50 years old; this fact sufficiently explains why fertility at 50+ is nil. But as the onset of menopause occurs at varying ages, the proportion of sterile couples inevitably increases with the age of the woman; the same is true of the proportion of women who become sterile by accident, as the result of a confinement, for example. The progression of secondary sterility,[7] whether it be natural or pathological, as the woman grows older is therefore a factor in the reduction of fertility. But this is not necessarily the only factor, for the fertility of couples who are still fecund can also be a reducing factor if this fertility decreases as the age of the woman rises.

[7]Secondary sterility, as opposed to "primary sterility," arises after one or more children have been born (cf. *Multilingual Demographic Dictionary*, published by the U.N., p. 38).

TABLE 1

AGE-SPECIFIC FEMALE LEGITIMATE FERTILITY RATES—RATES PER 1,000

Population	Age						Mean no. of children per completed family of woman married at 20	Mean fertility rate for 20-29 age group	Indices of mean fertility rates (base 100: mean rate 20-29)		
	20-24 years	25-29 years	30-34 years	35-39 years	40-44 years	45-49 years			20-29 years	30-39 years	40-49 years
Hutterites (3, 23) marriages from 1921-1930	550	502	447	406	222	61	10.9	526	100	81	27
Canada (10) marriages from 1700-1730	509	495	484	410	231	30	10.8	502	100	89	26
Hutterites marriages before 1921	475	451	425	374	205	29	9.8	463	100	86	25
Bourgeoisie of Geneva (12) wives of men born between 1600 and 1649	525	485	429	287	141	16	9.4	505	100	71	15.5
Europeans of Tunis (6, 16) (notabilities excluded) marriages from 1840-1859	468	430	402	324	190	13	9.15	449	100	81	22.5
Sotteville-lès-Rouen (Normandy) (8) marriages and births from 1760-1790	480	450	410	315	125	10	8.95	465	100	78	14.5
Crulai (Normandy) (7) marriages from 1674-1742	440	420	375	280	140	10	8.3	430	100	76	17
Norway (22) marriages from 1874-1876	396	380	341	289	180	41	8.1	388	100	81	28.5
Iran (villages) (11, 19) marriages from 1940-1950	395	370	325	255	130	20	7.5	382	100	76	19.5
Bourgeoisie of Geneva wives of men born before 1600	389	362	327	275	123	19	7.5	376	100	80	19
Taiwan (1, 15) (rural region of Yunlin) women born about 1900	365	334	306	263	114	8	6.95	350	100	81	17
India (18) (Hindu villages of Bengal) marriages from 1945-1946	323	288	282	212	100	33	6.2	306	100	80	22
Guinea (4) (villages of Fouta-Djalon) marriages from 1954-1955	357	320	273	183	74	32	6.2	339	100	67	16
Average	435	407	371	298	152	22	8.42	421	100	79.5	20.5
Great Britain (9) (women married at about 20 years old around 1920)	320	165	90	50	20	0	3.2				

Notes: 1. The numbers refer to the books and articles cited in the bibliography.
2. For Crulai and Sotteville-lès-Rouen, the above series of rates were obtained by graphically fitting the series published in the works cited.
3. For Norway, the rates including the still birth rates, given on p. 209 of the work cited, were multiplied by 0.96.

TABLE 2

COMPARISON BETWEEN THE AVERAGE OF AGE-SPECIFIC FERTILITY RATES FOR 13 POPULATIONS NOT PRACTICING BIRTH CONTROL AND RATES FOR A POPULATION PRACTICING CONTROL

Series of indices	20-24 years	25-29 years	30-34 years	35-39 years	40-44 years	45-49 years
Average series	100	93.5	85.3	68.7	34.9	5
Great Britain (Women married at about 20 yrs. old, around 1920)	100	51.6	28.1	15.6	6.2	0

TABLE 3

AGE-SPECIFIC PERCENTAGES OF STERILITY IN MARRIED WOMEN

Populations	20 years	25 years	30 years	35 years	40 years
England (middle of XIX century)	3	7	12	19	32
Rural Japan (pre-war)	4	10	19	33	53
Canada	3	4	6	10	22
Bourgeoisie of Geneva (wives of men born before 1650)	3	8	13	21	39
Europeans of Tunis	3	7	10	16	28
Crulai (Normandy)	2	3	7	15	36
Average (European populations)	3	6	10	16	31

The frequency of absolute sterility (primary and secondary) cannot be measured directly since the only observable phenomenon is infertility from a certain age. But because a married woman has been infertile since, let us say, the age of 40, it does not necessarily follow that she was sterile at 40; she has simply become so before having conceived again. Absolute sterility is less frequently found, therefore, than infertility. The former can only be measured from evidence of the latter, a method which is acceptable since sterility does not progress very quickly, at least not up to about the age of 40. From then on, one should proceed cautiously, fully aware of the risks of error entailed in such an undertaking.

Table 3 lists some approximate figures of the proportion of absolutely sterile couples according to the age of the woman.

All these different series show a low figure for women aged 20, 3% being the figure most often found, and also the over-all average. Then the percentages increase at varying rates of progression. But perhaps one should not attach too much importance to the differences between the European populations cited, as they can be attributed to errors in age, random fluctuations and the difficulties inherent in this type of calculation. The average series adequately depicts the general development.

The series for Japan is the only one that may be relevant for a non-European population. The rise in the sterility rates as the woman grows older is markedly steeper than for the other populations. The question arises as to whether this difference is peculiar to Japan or whether it would be found in other non-European populations. At present, it is impossible to answer this.

The proportion of fecund couples is the complement of the proportion of sterile couples; the product of the former multiplied by the fertility of the

fecund couples equals the fertility of the total of both groups.

Let us take as the percentage of fecund couples in one age group, the mean of the highest and lowest figures: 95.5 $\left(\frac{97+94}{2}\right)$ for the 20-24 age group, for example; the percentage for this group is then expressed as base 100 and the indices in Table 4 are obtained for the average of the European populations and for Japan.

These two series present the same general appearance; the reduction in percentage of fecund couples is accelerated with age, giving a sharply downward trend towards the oldest age group.

In order better to understand the significance of this outline, let us suppose for a moment that sterility were an accidental risk independent of age and affecting $s\%$ of all women confined. If, out of 1,000 women fecund at the beginning of the year, f gave birth in that year, there would only be 1,000 $(1\text{-}fs)$ fecund at the end of that year. Even if the value of f changes fairly gradually with age, the proportion of fecund married women would still be 1,000 $(1\text{-}fs)^2$ at the end of the following year, and so on. This series of proportions forms an outline which decreases as age increases during the first years and then flattens out, quite the contrary from what can actually be observed. Consequently the exposure to the risk of sterility caused by a confinement cannot be considered both important and independent of age. If the risk is considered an important factor, it must carry progressively more weight as age increases; alternatively, if the risk is considered independent of age, it is negligible.

The change in the proportion of fecund couples as the woman grows older is analogous to the change in the fertility rates of the total number of couples. The fertility rate of fecund couples is the other factor which affects the fertility rate of the total number of couples and which must now be examined. As this rate cannot be directly observed, one takes as a substitute the fertility rate of couples who later prove to be fertile, that is to say, fertile after the woman has exceeded the upper limit of the age group to which she belongs at the time of the survey (after the 30th birthday, for example, for women in the 25-29 age group).[8]

In each of the series (Table 5) there is a tendency for the figures to decrease as the age of the woman rises, though the rate of the reduction varies. Since the difference is at least partially due to sampling at random from a small number of cases, the mean rates give a better indication of the general picture.

The mean series of indices differs little from the series for percentage of fecund couples. Although the two series are not based on exactly the same groups of populations, the similarity does suggest that the decrease in the fertility of all the couples could be attributed to two factors of more or less equal importance: the reduction in the percentage of fecund couples and the reduction in the fertility of these couples.

The fertility rate of fecund couples is approximately equal to the inverse of the average of those intervals where the beginning or the end, or both, fall in the age group under consideration. The reduction in the fertility of fecund couples means that as the woman gets older so

[8]When calculating by age groups, the bias inherent in this method can be discounted up to at least the age of 40 as rates for couples who are later fertile are undoubtedly very similar to those for fecund couples.

TABLE 4
COMPARISONS BETWEEN EUROPEAN POPULATIONS AND JAPAN

Populations	20-24 years	25-29 years	30-34 years	35-39 years
European populations	100	96.5	91	80
Japan	100	92	79.5	61

TABLE 5
SOME SERIES OF AGE-SPECIFIC FERTILITY RATES FOR COUPLES WHO ARE LATER FERTILE

Populations		Rates per 1,000			
		20-24 years	25-29 years	30-34 years	35-39 years
Canada		522	519	517	495
Bourgeoisie of Geneva	1	552	542	514	397
Europeans of Tunis		495	476	433	381
Sotteville-lès-Rouen		533	479	449	331
Crulai (crude figures)		450	457	382	376
Bourgeoisie of Geneva	2	413	421	364	355
Mean rate		494	483	443	389
Mean index		100	97.5	89.5	78.5

births become more widely spaced. Thus, taking the mean series, the spacing goes from 24 months approximately for the 20-24 age group, to 25 months for the 25-29 age group, 27 months for the 30-34 age group, and 31 months for the 35-39 age group.

The same phenomenon can be observed within the family in a more abrupt form: the intervals between each child at first increase only gradually, but with the penultimate child and even more so with the last child, the gap becomes very much wider.

One can conclude from the progressive reduction in the fertility rates of fecund couples either that the wider spacing of children noted within the family takes place after the woman has passed the age of 40, or, and this is the more likely, that the ages where this lengthening of the interval takes place are sufficiently widely spread to annul the effect of the very rapid change noted within the family.

Let us now return to Table 1. The total of the fertility rates multiplied by 5 gives, approximately, the mean number of children per completed family of a woman married at exactly 20; a comparison between this number and the average fertility rate for the 20-29 age group (subtotal of the 20-24 and 25-29 age group rates) shows that the mean number of children per completed family of a woman married at 20 years old is twenty times greater than the mean fertility rate for the 20-29 age group. In other words, the number of children is the same as it would be if the level of fertility found among women of about 25 remained constant from 20 to 40 years, and then ceased completely after the 40th birthday.

The average number of children per completed family for women married at 45, 40, 35, 30, 25, and 20 years old can be obtained by adding the fertility rates in descending age group order and multiplying by 5. The average number of children thus calculated, using the mean of the 13 rates shown in Table 1, is an approximate linear function of the age at marriage when the age varies from 20 to 30, or even 35 years, as Table 6 demonstrates.

TABLE 6
NUMBER OF CHILDREN PER COMPLETED FAMILY BY AGE AT MARRIAGE OF THE WOMAN

	Exact age at marriage			
	20 years	25 years	30 years	35 years
Mean number of children	8.42	6.25	4.21	2.36

When plotted with age along the horizontal axis, the line expressing the average number of children declines to zero when the age at marriage is about 40. Thus an empirical result is reached analogous to the preceding conclusion: the mean number of children per completed family of a woman married between the ages of 20 and 35 equals the fertility rate for women of about 25, times the number of years between the age at marriage and 40 years.

So far, our efforts have been directed towards an exposition of the characteristics common to several series of legitimate fertility rates computed from populations not practicing birth control; now we must turn our attention to another essential fact: the level of fertility differs from one population to another. The highest level of fertility at about 25 years is 1.72 times greater than the lowest; the largest average number of children per completed family of a woman married at 20 is 1.76 times higher than the lowest average number. Thus, within the limits of the definition "natural fertility," and despite the similarity between one population and another in the pattern of legitimate fertility as it varies according to the age of the woman, the levels of fertility are very different. This can, of course, only be stated with qualifications, but as a simplified generalization it is useful for the purposes of examining the very important differences in level that have been established.

Before going any further, it would be well to anticipate the question of whether this difference really exists. The majority of figures relating to European populations, whether by origin or actual residence, and whether of modern or premodern societies, are subject to only minor errors; data on Taiwan are considered reliable thanks to the high standard of the system of registration instituted by the Japanese and to the checks carried out during the survey. The figures from Iran seem to be acceptable since, when applied to Muslim populations of the Mediterranean coast, they give birth rates which have been observed or evaluated by other means. The rates for India and Guinea are more open to question; the Indian series is undoubtedly characteristic of numerous surveys carried out there, but all the results seem to be flawed by more or less similar inaccuracies. The rates for Guinea are calculated from the births declared during the twelve months preceding the 1954-55 survey. But the average number of children per woman calculated from this series is considerably higher than the number stated by the women. On the other hand, as some of the children had just passed their first birthday, they had probably been counted as under one year old. There are then good reasons to believe that the rates observed are no lower than the real figures.

The differences observed, therefore, must be allowed to exist in fact, with the conclusion that there are very marked variations in fertility among popula-

tions *even in the absence of birth control.*[9]

Furthermore, nearly all the European populations observed show fertility rates that exceed those of the non-European populations. This result may seem surprising since the crude birth rate of non-European populations is in general higher than that of premodern European populations. But these rates, although very high, are still quite compatible with a legitimate fertility lower than that of premodern Europe, for among non-European populations fewer people remain single and the age at marriage is lower than among Western peoples; this is true both for the past and today. If this combination of high marriage rates and early age at marriage were accompanied by a legitimate fertility rate as high as that of premodern Europe, the birth rates would be raised even more and would reach figures that apparently have never been observed, even after correcting errors in registration.

Finally, the cause must be sought for these marked differences in fertility. *A priori,* it might be assumed that they are attributable to one of the two prime factors governing fertility: the proportion of fecund couples and the fertility of these couples.

The first can be quickly eliminated: the proportion of fecund couples where the woman is in her 20's varies only from 92% to 95.5% among the several European populations cited in Table 3. Japan admittedly shows a lower percentage, 88%, but the difference between this figure and those quoted for European populations is much slighter than the differences in the average fertility rates for the 20-29 age group.

The percentages by age of fecund women for the other populations is not known, but reliable figures are available showing the proportion of childless women who have reached or passed the age of menopause. It is 4% in Taiwan, for example, and about 7% for Fouta-Djalon. Considering the age at which women marry in these populations, figures such as these represent an upper limit to the frequency of sterility among couples where the woman is about 20 years old. But if 96% or even 93% of married women are fecund at 20, the percentage cannot drop to a figure as low as from 60%-70% for women of about 25; yet this low figure would be required to account for the differences observed in the fertility rates.

The differences are the consequence, then, of birth spacing; this is already apparent among European populations where the fertility rates of fecund couples, and consequently the mean intervals between births, show striking differences from population to population. For Taiwan, there is evidence pointing to intervals in the region of 2.7 years. In India, mean intervals in the region of 3 years have been found in surveys where the fertility rates are as high as those cited in Table 1.

We thus reach, whether directly or indirectly, the following conclusions.

First, marked differences in fertility do exist among populations where birth control is either unknown or very little practiced. As the incidence of absolute sterility is, at least among the younger age groups, similar from one population to another, the differences, for these younger groups, can be largely attributed to variations in the spacing of births by fertile couples.

[9]One European population with low natural fertility can moreover be cited—the Swedish parishes of Estonia studied by H. Hyrenius. This case does, however, appear to be an exception in Europe.

Second, although the differences appear among European populations, they are far more striking when European populations are compared with non-European; on the whole, the former space births far less than the latter.

The spacing of live births depends on several factors.

(1) The length of time that elapses between conception and either the resumption of sexual relations or the reappearance of ovulation, whichever occurs later after the confinement. This period can, though it is exceptional, be prolonged by temporary pathological sterility. A new conception is impossible during this "idle" period.

(2) Fecundability, or the probability of conception, outside the "idle" period defined above, measured by the menstrual cycle, or more simply by months.

(3) The frequency of miscarriages and still births.

It seems unlikely that the third factor can vary sufficiently to explain the differences that have been established. Rather, research on the populations of Geneva and Crulai seems to indicate that within one population the differences in the fertility of couples are largely due to variations in the length of the "idle" period. This finding has led us to formulate the hypothesis that differences between populations can be attributed to the same cause: that, in average, the resumption of sexual relations or the reappearance of ovulation occur at different times, later among some populations than others.

This hypothesis is borne out by the delay in the resumption of sexual relations imposed by the prohibition, among some populations, of relations during at least a great part of the lactatory period. These taboos are common in black Africa, but they do not exist among Muslim populations of the Mediterranean coast. In Europe, there were no such taboos, though traces can be found of popular beliefs which might have postponed the resumption of sexual relations; but the influence of these beliefs must have been considerably limited in numerous places by monogamy and the strictness of social mores.

The power of sexual taboos among the Africans is often quoted, and it seems to us rightly so, for it is known that they are very widespread in black Africa and it is generally considered that fertility is lower there than in any other region where birth control is not practiced. But, in fact, the taboos are not a complete explanation of fertility differences. On the one hand, they are unknown among certain African populations, the Ashanti of Ghana, for example, whose fertility rate, moreover, is very similar to that of other African populations. On the other hand, the problem is not simply one of the differences between Africa and the rest of the world, but of differences among many populations of which only some are African.

In order to maintain our hypothesis, then, we must also allow that the reappearance of ovulation after confinement occurs later among some populations than others. Since there is a relationship between ovulation and breast feeding, it might be thought that variation in the length of the suckling period from one population to another would account for the greater or lesser delay in the resumption of ovulation. But another explanation can be put forward: the relation between the lactatory and the anovulatory periods, whether of shorter or longer duration, is by no means constant. Ovulation is resumed very rapidly among some women even though they are suckling their babies.

With others some time elapses after confinement before it is resumed but still before the infant is weaned; and with still others, it is resumed only after weaning. In other words, women who breast feed for the same length of time can be divided into different categories according to the length of the anovulatory period. It is possible that the relative importance of each of these categories varies to a measurable degree from one population to another, and in these variations could be found at least a partial explanation for the differences noted in natural fertility.

Finally, the hypothesis by which populations may be differentiated according to the "idle" period, during which conception is impossible, leads us to distinguish between two explanations for the differences: first, differences in behavior relating to the resumption of sexual relations and the duration of the suckling period; and second, differences of a physiological nature relating to frequency and duration of anovulation during the suckling period.

To accept the second explanation is to admit that this physiological characteristic can be included among those which, for reasons of heredity or environment, vary from one population to another. Research in this field would help us to understand why there exist among populations such differences in natural fertility.

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PREDICTIONS IN FERTILITY*

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DURING THE PAST two decades there have been a number of field studies of factors associated with size of family. A few of these have collected data from both wives and husbands. In most cases the studies have been restricted to wives, largely for reasons of economy. It is apparent that full coverage of both partners not only doubles the number of questions to be asked but often necessitates the scheduling of evening appointments because the husband is away from home during the day.

Despite the practical importance of the problem, no studies have been directed toward ascertaining whether the relation of fertility to characteristics of the husband is the same or different from the relation of fertility to similar characteristics of the wife.

This paper attempts to examine whether fertility behavior can be predicted with the same degree of accuracy from the social and psychological characteristics of husbands as from those of wives. The major fertility studies in the past have related fertility behavior mainly to the characteristics of wives. The researchers of these studies tacitly assumed that the obtained results would have been the same if the characteristics of the husbands had been substituted for those of the wives. Freedman, Whelpton and Campbell (1959) have stated explicitly that some differences might have arisen in the results if data pertaining to husbands had been obtained directly from husbands instead of being supplied by the wives. Since the inclusion of husbands would have necessitated a substantial reduction of cases, these researchers decided to choose a larger sample restricted to the wives (Freedman *et al.*, 1959). Their decision probably would be endorsed by many demographers. Yet they leave unanswered the important question of whether reliance on information from only one spouse is sufficient. This is the essential question being investigated in this paper.

THE BACKGROUND OF THE STUDY

This paper is based on data collected for two different studies, the Kelly Study of marital happiness and the Indianapolis Study of fertility. The first data of the former study were collected by E. Lowell Kelly in the mid-thirties from 300 engaged couples, selected from announcements in eastern newspapers. Although Kelly was concerned with the study of marital happiness, the collected data also contained certain information regarding fertility behavior. The basic design of the study involved annual contact with the couples for an extended period of time. The advent of World

* This paper is based on the author's doctoral dissertation, *Predictors of Fertility*: A Comparative Analysis of Characteristics of Husbands and Wives Related to Fertility Behavior, submitted to the Department of Sociology, Graduate School of Arts and Science, New York University, 1960.

War II interrupted the plan, but in 1953-4 Kelly managed to learn the whereabouts of all the couples and to secure the desired data from a majority of them. Despite the fact that the data are not representative of the United States population (the engaged couples are largely restricted to the urban Northeast with about 65 per cent of them having some college education, and only about 10 per cent of them being Catholic), they are of unusual interest due to the longitudinal design of the study.

The second set of data used in this paper are from the Indianapolis Study. The purpose of that study was to examine whether fertility behavior could be predicted from the social and psychological characteristics of the couples. The data were collected in 1941 from a sample of 860 native-white Protestant couples married during 1927-9 living in Indianapolis, neither previously married, husband and wife having completed at least the eighth grade. The *ex post facto* design of the study complicated, to a certain extent, the interpretation of the results.

THE VARIABLES AND THE METHODS OF ANALYSIS

The dependent variables used in this paper are: (1) the total number of live births and (2) the size of completely planned families. A host of independent variables are considered in this analysis; the most important among them are: age at marriage, education, socioeconomic status, size of parental family, and liking for children.

Regression analysis was chosen as the most appropriate method. This method permits the introduction of factors which are related to the dependent variables with all of the other variables held constant. Of particular interest are the weights assigned to each independent variable by the regression equation. The coefficient for each independent variable in the equation is an index of the weight which the observations for that variable should be given in making estimates of the variance of the dependent variable. If these weights are expressed in standard units they are called beta coefficients. A value near zero for beta (either positive or negative) implies that the independent variable considered is of comparatively little importance in accounting for the variance of the dependent variable. These coefficients, like other statistical measures, are subject to sampling variations.

In order to select variables which have a high probability of influencing the dependent variable and not to select variables which have small weights as a consequence of sampling variability, a set of 95 per cent confidence limits was established for each beta. If zero falls within this specified confidence interval, then the variable must be suspected as having only a chance relationship with the dependent variable. Such procedure should help to establish which factors have high predictive values. For the Kelly data, confidence intervals for the betas were omitted because of the nonrandomness of the sample.

Table I contains the beta coefficients for the regression equations based on the characteristics of husbands and wives, respectively, from the Kelly data.

A comparison of the two regression equations in Table 1, one predicting total fertility from the characteristics of the husbands and the other from the corresponding characteristics of the wives, shows that net regression coefficients are, in most cases, approximately the same. Important exceptions are age at mar-

TABLE 1

COMPUTED VALUES OF BETA COEFFICIENTS FOR REGRESSION OF TOTAL FERTILITY UPON EIGHT CHARACTERISTICS OF HUSBANDS AND WIVES, AND THE REGRESSION EQUATIONS (KELLY STUDY).

Independent Variable	Symbol	Beta Coefficient	
		Husband*	Wife†
Age at Marriage	X_1	—.14	—.23
Education	X_2	—.19	—.03
Number of Children Desired	X_3	.17	.19
Size of Parental Family	X_4	.02	.23
Neurotic Tendency (Bernreuter‡)	X_5	—.02	—.05
Otis Test of Mental Ability‡	X_6	.09	.13
Social Adjustment Test (Bell‡)	X_7	.11	.13
Socio-Economic Status of Parents	X_8	.09	.04

*Regression Equation: $y = 1.94 - .06x_1 + .13x_2 + .18x_3 + .01x_4 - .05x_5 + .12x_6 + .13x_7 + .05x_8$.
†Regression Equation: $y = 1.15 - .12x_1 + .08x_2 + .19x_3 + .16x_4 - .11x_5 + .19x_6 + .07x_7 + .09x_8$.
‡For definitions and further details regarding tests, see Westoff *et al.* (1958).

TABLE 2

COMPUTED VALUES OF BETA COEFFICIENTS FOR REGRESSION OF COMPLETELY PLANNED FERTILITY UPON EIGHT CHARACTERISTICS OF HUSBANDS AND WIVES, AND THE REGRESSION EQUATION (KELLY STUDY).

Independent Variable	Symbol	Beta Coefficient	
		Husband*	Wife†
Age at Marriage	X_1	—.02	—.18
Number of Children Desired	X_3	.24	.32
Size of Parental Family	X_4	.12	.26
Neurotic Tendency (Bernreuter)	X_5	—.03	—.01
Childhood Happiness	X_9	.09	.14
Frequency of Home Discipline	X_{10}	—.03	—.14
Optimism	X_{11}	.11	.19
Extra-Curricular School Activities	X_{12}	.19	.23

*Regression Equation: $y = .91 - .01x_1 + .25x_3 + .07x_4 - .02x_5 + .09x_9 - .05x_{10} + .10x_{11} + .16x_{12}$.
†Regression Equation: $y = 1.51 - .07x_1 + .30x_3 + .19x_4 - .01x_5 + .14x_9 - .18x_{10} + .14x_{11} + .20x_{12}$.

TABLE 3

COMPUTED VALUES OF BETA COEFFICIENTS FOR REGRESSION OF TOTAL FERTILITY UPON NINE CHARACTERISTICS OF HUSBANDS AND WIVES, AND THE REGRESSION EQUATIONS (INDIANAPOLIS STUDY).

Independent Variable	Symbol	Beta Coefficient		Factors Significantly Different From Zero at 5 Per Cent Level?	
		Husband*	Wife†	Husband*	Wife†
Age at Marriage	X_1	—.15	—.19	Yes	Yes
Education	X_2	—.09	—.11	Yes	Yes
Number of Children Desired	X_3	.27	.29	Yes	Yes
Number of Biological Sibs	X_4	.01	.03	No	No
Marital Happiness‡	X_{13}	—.11	—.06	Yes	Possibly
Index of Liking for Children	X_{14}	.15	.14	Yes	Yes
Index of General Planning	X_{15}	—.10	—.06	Yes	Possibly
Index of Personal Adequacy	X_{16}	—.02	—.04	No	No
Conformity Index‡	X_{17}	—.02	—.04	No	No

*Regression Equation: $y = 2.19 - .08x_1 - .05x_2 + .25x_3 + .01x_4 - .07x_{13} + .14x_{14} - .16x_{15} - .02x_{16} - .01x_{17}$.
†Regression Equation: $y = 2.09 - .11x_1 - .08x_2 + .28x_3 + .02x_4 - .04x_{13} + .15x_{14} - .09x_{15} - .05x_{16} - .03x_{17}$.

‡The indices in Tables 3 and 4 were retained in the original form in which they were constructed and used by various authors of the Indianapolis Study. For details regarding construction of indices see Whelpton and Kiser, 1950, 1952, 1954, 1958.

TABLE 4

COMPUTED VALUES OF BETA COEFFICIENTS FOR REGRESSION OF COMPLETELY PLANNED FERTILITY UPON TEN CHARACTERISTICS OF HUSBANDS AND WIVES, AND THE REGRESSION EQUATIONS (INDIANAPOLIS STUDY).

Independent Variable	Symbol	Beta Coefficient		Factors Significantly Different From Zero at 5 Per Cent Level?	
		Husband*	Wife†	Husband*	Wife†
Age at Marriage	X_1	—.11	—.17	No	Yes
Education	X_2	.16	.03	Yes	No
Number of Children Desired	X_3	.25	.21	Yes	Yes
Number of Biological Sibs	X_4	—.06	.01	No	No
Marital Happiness	X_{12}	—.07	.06	No	No
Index of Liking for Children	X_{14}	.36	.24	Yes	Yes
Index of General Planning	X_{15}	—.12	.08	No	No
Index of Personal Adequacy	X_{16}	.01	—.13	No	No
Index of Interest in Religion	X_{18}	—.03	—.07	No	No
Index of Economic Security	X_{19}	.19	.28	Yes	Yes

*Regression Equation: $y = 1.69 - .05x_1 + .06x_2 + .19x_3 - .02x_4 - .03x_{12} + .20x_{14} - .13x_{15} - .01x_{16} - .02x_{18} + .25x_{19}$.

†Regression Equation: $y = 1.28 - .07x_1 + .01x_2 + .15x_3 + .01x_4 + .01x_{12} + .15x_{14} + .08x_{15} + .10x_{16} - .06x_{18} + .36x_{19}$.

riage, size of parental family, and education. The regression coefficients for the first two variables are higher for the wives than for the husbands, but the coefficient for education is higher for the husbands than for the wives.

Attention may now be turned to the relation of characteristics of husbands and wives to *completely planned* fertility. Table 2 presents the beta coefficients and the corresponding regression equations.

As indicated by Table 2, the net regression equations based on the characteristics of wives tend to be larger than those based on the corresponding characteristics of husbands. Thus, in this study the characteristics of wives are better predictors of completely planned fertility than those of the husbands. Furthermore, from the Kelly data, the results with regard to total fertility are incongruous with those relating to completely planned fertility. Since the data are from a highly selected sample, caution must be used against generalizations beyond the data.

Next will be examined the relationships of the characteristics of husbands and wives from the Indianapolis Study to their total fertility and to their completely planned fertility. The beta coefficients based on certain characteristics of husbands and wives and the regression equations are presented in Table 3.

Table 3 shows that, in general, the factors significantly different from zero for the wives are also significantly different from zero for the husbands. However, two factors—index of general planning and marital happiness of wives—have a somewhat weaker relationship to total fertility than the corresponding factors of husbands. In general, total fertility can be predicted with approximately the same accuracy from the given characteristics of husbands as from the same characteristics of wives. Between the data from husbands and those from wives, there is some difference in the magnitude of the net regression coefficients, but it is not large enough to be of importance.

The relationships between the characteristics of husbands and wives and completely planned fertility are presented in Table 4.

Table 4 indicates that the beta values for four characteristics of husbands and wives used for prediction of completely

planned fertility are significantly different from zero at the 5 per cent level. Three of these characteristics (number of children desired, index of liking for children, and index of economic security) have beta values significantly different from zero at the 5 per cent level for both husbands and wives, although the values are somewhat larger for husbands. The factor of education has a much higher predictive value for wives than for husbands insofar as size of completely planned family is concerned.

Both the Kelly and the Indianapolis Studies show that total fertility can be predicted with approximately the same accuracy from the characteristics of husbands as from those of wives. In contrast, the predictive values of the characteristics of husbands are different from those of the wives for completely planned fertility. The lack of congruity of the results in the latter case is probably due to sampling fluctuations. Due to certain shortcomings of the data of both studies, conclusive generalizations cannot be made. However, the results obtained suggest that predictions in fertility studies should concentrate on completely planned fertility. This type of fertility behavior is important also because, in the near future, planning of family size will become increasingly important in countries of Western civilization.

SUMMARY

The test of the hypothesis that fertility behavior can be predicted from the social and psychological factors of either spouse was the subject of this paper. The data of the Kelly and the Indianapolis Studies were used for testing the hypothesis. The method used was that of regression analysis. These data supported the hypothesis with respect to total fertility but not for completely planned fertility. Since both sets of data suffer from certain shortcomings, further testing of the hypothesis is necessary for reaching conclusive results.

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Brief Reports and Communications

THE CONNECTICUT TWIN REGISTRY

The Connecticut State Department of Health is establishing the Connecticut Twin Registry. A listing of all multiple births occurring in the State of Connecticut since January 1, 1897, is being compiled. The birth records of all the local Registrars of Vital Statistics are being searched for multiple births occurring between 1897-1955. A duplicate set of punch cards on all multiple births occurring since January 1, 1956, is being furnished annually by the Public Health Statistics Section of the State Department of Health.

All multiple births are checked against death certificates. Only those multiple births in which at least two members survived to one year of age have identifying information punched on IBM cards. Information concerning perinatal, neonatal, and infant mortality is being collected but this information is not routinely placed on punch cards.

The Registry is being created to obtain a twin population to use in conjunction with the Connecticut Tumor Registry for genetic studies of human cancer. The Registry is also intended to be used as a research source for studies of disease and studies in the fields of genetics, psychology, sociology, etc. The Registry will cooperate with trained individuals who wish to use its facilities for research purposes, and will supply available data to them.

Researchers requesting data of the Connecticut Twin Registry will be expected to abide by such requirements as may be set forth by the State Department of Health. All requests will be reviewed as to the qualifications of the investigator and to the merit of the proposed study. In order to avoid duplication of efforts, abstracts of information obtained and zygosity determinations will be requested of investigators using these facilities.

Requests for further information and/or use of the listings of multiple births should be made to:

MERTON S. HONEYMAN
Geneticist
Chronic Disease Control Section
Connecticut State Department
of Health
Hartford, Connecticut

A SHORT COURSE IN MEDICAL GENETICS

JOHN L. FULLER AND VICTOR A. MCKUSICK

The Roscoe B. Jackson Memorial Laboratory and
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GENETICS HAS recently assumed a more important place in the medical curriculum. This is in part because conquests in the areas of infectious disease and nutritional disease have made diseases of genetic origin relatively more conspicuous in our population. The genetic consequences of radiation exposure also loom high as public health hazards. Another reason for increasing interest in medical genetics has been the noteworthy discovery that many inherited diseases are actually molecular diseases whose origin is to be sought in faulty construction of basic cell constituents. Variations in the hemoglobin molecule produce conditions such as sickle cell anemia, a serious health problem in some regions of the world. Furthermore, important advances have been made in relating chromosomal aberrations to clinical syndromes. Perhaps most noteworthy has been the finding that mongolism, a type of mental deficiency whose origin was long shrouded in mystery, can be attributed to an extra autosomal chromosome which disturbs metabolic balance in the organism.

The introduction of any new discipline to the medical curriculum presents grave difficulties when it must be added to an already crowded list of requirements. Furthermore, keeping up-to-date in a rapidly moving area of science is difficult for an individual working by himself. To meet the needs of various categories of individuals who wished an introduction to the field, the short course in Medical Genetics was conceived in the summer of 1959. As originally outlined, the purpose of this course was to provide for faculty members in medical schools, universities and certain other professional schools an opportunity to become acquainted with the recent work in a broad area covered by the term "Medical Genetics." Thus the course was to include much more than the inheritance of specific disease conditions. An attempt would be made to provide insight into the development of modern ideas of heredity and to integrate medical applications with pertinent aspects of experimental mammalian genetics. This objective was emphasized by the subtitle chosen for the course, "Heredity and Disease in Man and Animals." To staff such a program required the collaboration of a medical school faculty and a staff which was primarily concerned with the basic aspects of mammalian genetics. Discussions were initiated between representatives of the Johns Hopkins University School of Medicine and the Roscoe B. Jackson Memorial Laboratory. As a result of preliminary discussions, the staffs of these two institutions decided to pool their resources and to present in the summer of 1960 an intensive two-week course on the principles of medical genetics.

On the basis of preliminary plans developed at Johns Hopkins and the Jack-

son Laboratory, a proposal was submitted to the National Foundation for financial support to cover staff costs, teaching expenses and the rental of suitable quarters. The Foundation responded generously, making it possible to eliminate completely all tuition fees, and we are happy to record that similar support has been granted for 1961. The Course was conducted at Bar Harbor, Maine, in the Oakes Center of Bowdoin College, a beautiful mansion located on the shores of Frenchman's Bay. The pleasant and restful surroundings were a major contribution to the success of the venture since distractions were at a minimum and participants could devote full time to reading and discussion.

Announcement of the course was made through the scientific press early in 1960. Originally it was planned to take 30 students, but the number of acceptable candidates increased so that this quota was raised, first to 40 and then to 45. Actually over 200 completed applications were received and many well-qualified individuals could not be accepted because of limitations of space. The final group included representatives from medical schools in 19 states and provinces, one dental school and two schools of veterinary medicine. The academic status of the "students" ranged from Deans of Faculties to recent graduates holding hospital residencies.

As finally constituted, the teaching staff included nine representatives from the Johns Hopkins University and 11 from the Jackson Memorial Laboratory. Eight visiting lecturers also contributed one or more lectures each. Classes met each morning for four hours and again for a two-hour session in the evening. Afternoons were left free for recreational activity, but a number of the participants used this period for reading in the small library set up especially for the course, while others were regular attendants at a series of films illustrating major principles of genetics.

The first week was devoted essentially to basic genetics, starting with mitosis and the formation of germ cell, extending through blood groups, biochemical genetics, and cytogenetics and into the statistical aspects of genetic investigations. Considerable emphasis was given to the pitfalls of family investigations, and the fact that simple recurrence of a disorder within a family is not enough arbitrarily to label it as hereditary. The second week dealt more specifically with individual syndromes and classes of disorders. The subjects ranged from muscular dystrophy, congenital malformations, anemia, and neurological defects to cancer, circulatory disorders, psychiatric conditions and radiation genetics. Obviously, it was impossible in a short period to deal exhaustively with any of these subjects, but an effort was made to bring the student body up-to-date on the current situation in each class of conditions. The curriculum for 1961 has been revised on the basis of the 1960 experience. Table 1 provides the tentative schedule for 1961.

Staff and students both seemed to enjoy themselves during the two-week session. At the end of the period all individuals were invited to submit a critique of the course, and favorable comments outweighed the negative ones. However, on the basis of these statements, modifications in the curriculum are planned for the year 1961. In general, the revised course will devote somewhat more time to basic genetics, including the nature of the gene, and the relation of nucleic acid structure to the transmission of genetic information. To compen-

TABLE 1
SHORT COURSE IN MEDICAL GENETICS

Date	Morning	Evening
Monday, August 7	Principles of genetics: I. Segregation and independent assortment. II. Linkage; inferences from breeding experiments.	Physical basis of heredity. I. Mitosis, meiosis, crossing-over.
Tuesday, August 8	Physical basis of heredity. II. Chromosomal aberrations. Allelism. Pedigree patterns.	Cytogenetics in man.
Wednesday, August 9	The chemical basis of inheritance.	The variant hemoglobins.
Thursday, August 10	Chemical aspects of gene action. I. Introduction. II. Genetic polymorphisms of serum proteins.	Tryptophane synthetase, a model of biochemical genetics.
Friday, August 11	Chemical aspects of gene action. III. Inborn errors of metabolism.	Experimental analysis of gene action, with particular reference to the mouse.
Saturday, August 12	Gene action: The blood groups.	
Monday, August 14	Gene action and immunity. I. Biologic individuality. II. Histocompatibility. III. Resistance to infection.	Biology of mutation I.
Tuesday, August 15	Genes in populations. I. Equilibrium states. II. Factors influencing gene frequency. Biology of mutation II.	Quantitative inheritance.
Wednesday, August 16	Genes in kindreds (Statistical methods, ascertainment biases, modes of inheritance, linkage).	Panel: Selection and evolution in man.
Thursday, August 17	Genes in development.	Congenital malformations.
Friday, August 18	12 short invitation papers presenting recent works which illustrate approaches and principles in medical genetics.	

sate there will be a reduction in the amount of time devoted to the discussion of specific familial diseases, though enough will be given to illustrate the principles of hereditary disease in human populations.

As originally conceived, the course had the particular objective of bringing medical faculties up-to-date information which might be introduced into existing courses. It was recognized that really satisfactory teaching of medical genetics requires professional geneticists attached to the staff and that a short course cannot provide this type of training. Exposure of pediatricians and internists to geneticists seemed to strengthen sentiment for the introduction of such programs into medical schools. In several instances the short course seemed to provide an added stimulus for participants to continue their training in genetics. At least three members of the first class will be affiliated in 1961 with medical genetics centers in leading medical schools for the purpose of obtaining advanced training in this area. Thus, recruitment of personnel has been added

to the original objectives, particularly for those participants who are just beginning their professional careers. It is hoped, also, that acquaintance on the part of deans and senior faculty of medical schools with the scope of, and opportunities in, medical genetics will lead to their counseling promising young men to enter this area. Still another objective is to provide research workers in the medical sciences with information on recent advances in genetics which may further their investigations, and to acquaint them with useful models from the field of mammalian genetics. Although medical genetics will always be predominately human genetics, there are many analogues of human diseases in mice, rabbits and dogs which can be studied experimentally with great profit. The close contact of medical scientists with individuals working predominately with animals is bound to produce significant interactions at the staff level as well as among the students. Because the course is provided with a faculty of liberal size and wide competence, all of these objectives can be approached simultaneously.

Perhaps stimulating intellectual interaction among the faculty should be listed as an extra dividend, one which may become progressively more important in later years. Opportunity for the interchange of ideas was used to the full and several cooperative research projects were initiated in the coffee hours held on a terrace overlooking Frenchman's Bay.

Plans are proceeding for the 1961 course and the number of applications again suggests that the demand for such training far exceeds our capacity to provide it. We hope to repeat the course annually until the backlog of demand has been substantially reduced, and perhaps thereafter offer such training at regular intervals as a refresher for past students and as an opportunity for younger individuals considering the field of medical genetics as a career. It is too early, of course, to know all the repercussions of this program, but its success must be measured by the extent to which (1) it stimulates individuals to become students of medical genetics, (2) it raises the level of the teaching of medical genetics in medical schools, and (3) it provides communication between the clinical branches and the research workers who have an intimate concern with problems of heredity and disease.

Book Reviews

GENETIC BASIS OF MORPHOLOGICAL VARIATION: Richard H. Osborne and Frances V. DeGeorge. Harvard University Press, Cambridge, 1959, 191 pp., $6.00.

The somewhat naive separation of Nature and Nurture into neat pigeonholes is no longer acceptable. As Professor Dobzhansky emphasizes in the excellent Foreword to this book, "All traits and all characters are hereditary and all are environmental." What we wish to know, then, is how much of the variance found in a particular pattern of development in a group of humans is influenced by the variety of genotypes present; and how much is due to the diversity of environmental factors.

The authors have studied a group of "healthy" adult twins to determine this interaction, and the distribution of these influences. They investigated the morphology of the sample—both by measurements and description. The book describes the methods and results of a statistical analysis of these data. They used the principle of estimating and comparing the ratios of the variances between the individual twins of monozygous pairs and dizygous pairs. These variances were also compared to the variances between the pairs themselves.

Carefully collected anthropometric data were analyzed together with data on fat, bone and muscle status. Body-build data and method were less satisfactory but provocative, and a good method of describing morphological bisexuality, applicable to both sexes, completed the over-all picture.

From the results of this analysis some good points are made. The magnitude of within-pair differences in twins is of a quite different order than that found, for example, in studies on unrelated individuals. The analysis suitable for twin studies is most exacting and failure to find significant differences in measurements of twins does not preclude similar differences being very useful in other types of studies for describing, say, environmental influences. In contrast to studies on birth weight and weight in children, no such measurable genetic component of variability could be found in these adult twins. Stature, on the other hand, showed a measurable genetic component. Whereas practically all anthropometric measures in childhood made on twins have demonstrated significant genetic influence (growth rates and patterns seem to be largely genetic), in this adult study some measures showed measurable genetic variability in one sex but not in the other. The fat, bone, and muscle estimations all showed a significant genetic component of variability, as did the "bisexuality" ratings.

The sample sizes are somewhat small, though larger than usually found in past studies (and perhaps the review of other studies could have been a little more comprehensive). However, the authors explain that they set out in the hope that, if their findings were positive and meaningful, they would point the way for better methods and more detailed twin studies in this area. Multi-variate analysis would be one valuable tool that

springs to mind. While nothing completely new has been presented, the collection, analysis and exposition of their data is a valuable and stimulating addition to the genetic storeroom.

The presentation is orderly, easy to absorb and the ninety tables are clear. The question does come to mind as to whether this careful study is best presented as a book. The price is steep.

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Child Development Unit
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THE RICH AND THE POOR; A STUDY OF THE ECONOMICS OF RISING EXPECTATIONS: Robert Theobald. A Mentor Book, New American Library, New York, 1961, 160 pp., 50¢ (first published by Clarkson N. Potter, 1960).

THE CHALLENGE OF ABUNDANCE: Robert Theobald. Clarkson N. Potter, New York, 1961, 235 pp., $4.50.

"Economics," a famous practitioner of the art once wrote, "is what economists do." These two recent books by Robert Theobald, an economic consultant working for such organizations as the American Management Association, General Electric and the Economist Intelligence Unit, illustrate that the boundaries of economics continue to be highly elastic. Mr. Theobald's underlying concern in both works is that too many of our ideas are based upon neoclassical economic doctrines (or "conventional wisdom," as Galbraith called it), whose assumptions no longer fit the facts of the modern world. Rejecting this inherited suit of theoretical clothes, he goes forth instead in a patchwork of garments borrowed from political scientists, anthropologists, sociologists, *et al.* His relative nakedness may not in fact prove so shocking to other economists as he seems to suggest, since much modern writing on economic problems has recognized the need for a synthesis of the approaches of the various social sciences. But the economist—particularly the practical economist who has to find specific answers to specific problems—may complain with some justice that Mr. Theobald's approach does not deliver "a new theory of global economics" or a "blueprint for future policy," as claimed on the publishers' jackets.

In any event, Mr. Theobald is not writing for economists. His analysis is directed at the reader who wants a stimulating and readable discussion of the major world economic problems of our time without technical jargon. This he does deliver admirably. *The Rich and the Poor* (now available also in paperback from Mentor Books) surveys in broad socioeconomic terms the dilemmas of the poor countries trying to become rich, and of the rich countries wondering what to do with their riches once they have them. The enormous and many-faceted complexity of these problems is well described. *The Challenge of Abundance* concentrates more on the problems of the rich countries, particularly those of affluent America. It is difficult here to avoid comparison with Galbraith's *The Affluent Society*, which covered much the same ground and came to many of the same conclusions. Both books end on the note that the rich countries must, for reasons of self-interest as well as from moral obligation, develop more effective means of aiding the poor.

The economist is bound to return to the criticism that the socioeconomic approach (for want of a better word) is much more successful in stating the challenges than it is in suggesting how

to meet them. The solutions proposed by Mr. Theobald have in general a tendency to veer from the practical to the ethical, boiling down to the indisputable proposition that the world would be a better place if unselfish enlightenment were to reign supreme among the arbiters of economic affairs. However, such a criticism should not be allowed to detract from appreciation of the value of a broad and lucid explanation of the problems involved. For, as the author recognizes, it is only through understanding the problems that any solutions will ever be found.

F. D'A. COLLINGS
Washington, D. C.

THE HAZARDS TO MAN OF NUCLEAR AND ALLIED RADIATIONS: A Second Report to the Medical Research Council, Her Majesty's Stationery Office, London, 1960, 154 pp., price 7s. 0d.

This report follows the same outline as the first one published in 1956 (and reviewed in *Eugenics Quarterly*, March 1957). Recent knowledge is presented and interpreted in six chapters, the last of which (Assessment of the Hazards of Exposure to Radiation) forms an excellent and concise summary. A more detailed discussion of data from research in the United Kingdom concerning selected problems is found in eleven appendices.

A helpful feature is the frequent comparison of current data and interpretations with those in the 1956 report. The relevant data are now more precise, and there are better procedures for detecting and limiting exposure to radiation. The suggested "permissible" levels of radiation are essentially the same as before, while some of the hazards now appear less dangerous than before.

One portion of the report deals with the effects of radiation. Relatively little new information is presented concerning the *early* effects (those developing within a few weeks or months). Among the *delayed* effects, knowledge is now most adequate about leukemia. Epidemiological data and experiments in the mouse are reviewed, with the conclusion that the risk of leukemia following radiation may be somewhat less than was assumed in 1956. Recent evidence suggests that the *genetic* effects probably depend upon the level and rate of dosage, in addition to the total dose accumulated. The concept of a "doubling dose" of radiation is now seen to be meaningful only when the analysis specifies factors such as the stage of development of the germ cells, the type of radiation, the rate at which it is received, and the age and sex of the recipient. We still know little about the role of radiation in causing chromosomal changes of the type recently described in man.

Concerning the sources of radiation, the report points out that man has been accustomed for long ages to natural radiation as part of the environment. The average dose to the gonads from the natural background is estimated at 85 to 106 millirads per year. Data are presented to show the variation in gamma-ray dose rates in Great Britain, particularly as related to the type of rock used in house construction. The total occupational dose is estimated at 0.4 per cent of that from the natural background. The dose from all kinds of medical radiology is estimated at 19.3 millirads annual genetically effective dose. The committee feels that this medical exposure could be reduced to 6 mrads with no curtailment of the benefits if greater consideration were given to improvements in technique. The additional dose

rate from radioactive fallout is slightly more than one per cent of that from the natural background.

The decision as to permissible doses of radiation must be based upon concern for individuals as well as for the general population. Maximum levels are thus suggested for three groups of persons: (1) those occupationally exposed to radiation, (2) those not directly exposed but living in the vicinity of areas requiring radiological supervision, and (3) the general population. Special rules have been set up as a guide for protecting the general public following accidents in nuclear reactors. Data from the investigation of such an accident in 1957 at Windscale are presented in considerable detail.

Of special interest to this reviewer was a discussion of the passage of radioactive debris from residence in the stratosphere to the troposphere and to the ground via rain, the mechanisms of plant contamination, the portals of entry into the human body, and the resulting radiation doses to the tissues. Analyses of human bones for strontium 90 have shown the highest level in infants between a few months and a few years of life. The level of strontium 90 in the diet depends more upon the material deposited recently by rain than upon the accumulated total in the soil. The fallout of artificial carbon 14 is also considered, with the suggestion that gene mutations may be produced by the disintegration of carbon 14 atoms which have been incorporated into DNA molecules.

The report also presents an estimate of what would happen if test nuclear explosions were to continue indefinitely with the same fallout pattern as for the period May 1958 to April 1959. The calculations, of course, are limited to the present knowledge of the contributing factors, and they are further limited to data from the United Kingdom. With these assumptions it is shown that the radiation from fallout would eventually reach an equilibrium at which the dose rate to the gonads would be about one-seventh of that from the natural background, while the dose to bone would be one-third and the dose to bone marrow one-fifth of that from the natural background. The committee, however, restated the 1956 conclusion that "atomic warfare on a large scale could not fail to increase for many generations the load of distress and suffering that individuals and all human societies would be called upon to support."

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Race and Reason. A Yankee View: Carleton Putnam. Public Affairs Press, Washington, 1961, 125 pp., $3.25.

This book is a collection of the more respectable arguments commonly used to justify racial segregation. It appears to be written with sincere conviction and, for the layman, with dangerous persuasiveness. Although he addresses primarily those Northerners who are somewhat fearful of integration, Putnam evidently realizes that his book will be read more widely in the South; accordingly, he includes advice for Southerners as well as arguments for Northerners.

The first two chapters are somewhat autobiographical and explain that the author is a lawyer-businessman who has lived in the South as well as in the North. These chapters reprint the two "Putnam letters" addressed respectively to President Eisenhower in October, 1958, and to Attorney General Rogers in

March, 1959, which were published widely in Southern newspapers; the first also appeared as a paid advertisement in Northern papers. The first letter mainly questioned the justice and the wisdom of enforced integration. The second attempted to show that the Supreme Court decision on segregation in 1954 was based largely upon erroneous scientific claims of racial equality.

The body of this small book is a series of questions and answers that purportedly grew out of correspondence over the two published letters. The questions are fairly representative of the controversial issues, and a few of them are answered well. Putnam has tried to bring reason to bear on nearly every question, but he also takes occasion to discredit the proponents of racial equality by mentioning their alien origins or their alleged control by an "equalitarian conspiracy." The facts and the logic are not very sound scientifically, and this reviewer finds many points of disagreement with Putnam's "genetic truth."

From a detached scientific point of view, the author's most grave and pervasive error is his failure to analyze the race problem in terms of individual human beings. This is emphasized by a footnote on page 36: "While scientists dislike the concept suggested by the word 'average,' preferring the concept of 'frequency,' I have used 'average' throughout this book as more understandable to the layman and equally acceptable in the context in which I employ it."

A scientist uses averages in some contexts, frequencies in others, and the two are rarely if ever interchangeable. It is doubtless true that use of frequency concepts, where appropriate, would have impeded communication between the author and his audience. This is partly because the author and most laymen habitually think in terms of "the average Negro" and partly because many of the arguments vanish into thin air when properly phrased in terms of trait frequencies. If Putnam wishes to challenge scientists in their own discipline, he should not begin by abjuring valid and precise scientific language.

The author defends his preoccupation with "the average Negro" on pages 28-29: "Any American worthy of the name feels an obligation of kindness and justice toward his fellow man. He is willing to give every individual his chance, whatever his race, but in those circumstances where a race must be dealt with as a race, he realizes that the level of the average must be controlling, . . ."

The premise that Negroes must be dealt with "as a race" is ostensibly based on genetic concepts to be discussed below. But implicit in much of this book is another, probably more primary reason: in the South, institutions are *designed* to deal with Negroes as a race. Thus the existence of these institutions is woven, perhaps unwittingly, into the fabric of the argument with which they are defended.

The alleged scientific justification for dealing with Negroes as a race stems from a mistaken notion of the genetic nature of individual and racial differences (p. 42): ". . . in those matters which involve social association, and hence the possibility of intermarrying, the element of race inevitably enters because each individual carries in his genes the heritage of his race and this will be passed on in the breeding process." Putnam evidently regards races as absolute and nearly homogeneous divisions of mankind, instead of the arbitrary classifications on a continuum that they are. Ignoring the particulate nature of hered-

ity, Putnam infers that each individual carries the genetic qualities of his entire race and that all his genes are, in a sense, marked. The facts indicate that, for the most part, Whites and Negroes share the same genes and differ mainly in the relative frequencies of these genes. Each individual has his own assortment of genes, his "genotype." With respect to the sort of hereditary variations that might influence adaptation to civilization, an individual's manifest traits are the best guide to his genotype. These may be a poor guide, but the traits of preceding generations are even poorer and remote ancestry can add no information at all. Thus, even when intermarriage is in question, science demands that each person be considered on his own merits.

Pursuing the subject of intermarriage further, Putnam asserts that Negro-White marriages would inevitably "pull down" the White race. Even if we grant, for the moment, the author's contention that Negroes are on the average genetically less adaptable to civilization, marriages are contracted between individuals, not between races. If, as Putnam admits, many individual Negroes may be "superior" to many individual Whites, the long-term outcome of scattered interracial marriages will depend on which Negroes marry which Whites.

Finally, we must examine the vexed question of the "average" Negro's relative adaptability to civilization. Some inconsistencies in Putnam's argument must be passed over if his main point is to stand out clearly. About 4000 years ago early civilization started to spread from Iraq and Syria into Asia and southern Europe, but not from Egypt to the rest of Africa. This Putnam attributes to relatively low receptivity to civilization on the part of African Negroes. Even if accidents of climate and geography could be ruled out, and they cannot, other weaknesses of this argument deserve closer examination.

By its nature as a sort of feed-back or resonating system, human culture is apparently capable of immense, if inexact, amplification of very small primary variations between human populations, whether of environmental or genetic origin. Hence, the wide contrast between modern European culture and the culture of Africa, even if due to an initial difference in adaptability to civilization, is not as Putnam asserts, a *measure* of that difference. An extremely small average difference between the Whites and Negroes, meaning a slightly higher frequency of receptive *individuals*, would suffice to explain the northward movement of civilization, if other circumstances could not be found to account for it.

In conclusion, this is a book to beware of. The author makes some valid points, but these are lost among his errors. The four scientists who have signed the introduction (Gates, Garrett, Gayre and George!) are at fault not for speaking out on the side of racial segregation, but for attaching their names, as scientists, to a book that confessedly and repeatedly violates scientific standards of accuracy.

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MEETINGS AND RECENT PUBLICATIONS

DIABETICS UNKNOWN: Groff Conklin. Public Affairs Pamphlet No. 312, Public Affairs Committee, Inc., New York, 1961, 27 pp., 25¢ (paper).

This pamphlet stresses that about half the people in this country with diabetes don't know they have the disease. It touches upon the role of heredity in diabetes, stating very properly the very grave risks for developing the disease if both one's parents are diabetic. However, some heredity counselors may differ with other risk figures given in certain family relationships. Geneticists would agree with the precautions suggested for those with a family history of diabetes: regular medical advice for possible avoidance of the condition and investigation of a possible diabetic background in a prospective marriage partner.

The pamphlet supplies useful information for detection and treatment of diabetes and outlines the great strides made in control of the disease which give diabetic women good chances for having children. The need for better research is very great, concludes the author.

It is encouraging to have the hereditary basis of a widespread disease discussed so frankly in a popular pamphlet. Organizations and individual writers have not always been willing to publicize inherited factors in common diseases because of the mistaken belief that if it's hereditary it's incurable. The future of preventive medicine may very well lie with some of the so-called "inherited" diseases, according to some outstanding geneticists today.

HUMAN GENETICS—A New Service for Minnesota: Human Genetics Unit, Division of Special Services. Minnesota Department of Health, Minneapolis, 1961, 4 pp. (paper).

This leaflet describes the pioneering services in human genetics of the Minnesota Department of Health which may prove to be of great significance in the history of public health in this country. In 1959 the Minnesota State Legislature authorized the Minnesota Department of Health to establish a human genetics program and appoint an advisory committee. The Dight Institute of Human Genetics, one of the oldest American heredity counseling centers, and the Minnesota Human Genetics League supported this legislation. Sheldon Reed, Director of the Dight Institute, is chairman of the advisory committee.

This public health program provides for both genetic counseling and research. It replaces the consultation services formerly provided by the Dight Institute at the University of Minnesota. The Human Genetics Unit works with physicians and dentists; private hospitals, institutions, and agencies; official state and local institutions; individuals by referral from physicians or dentists. Only general information will be supplied to individuals without referral. Requests for services should be directed to the Human Genetics Unit, Minnesota Department of Health, University Campus, Minneapolis 14, Minnesota.

CRIME IN AMERICA: Edited by Herbert A. Block. Philosophical Library, New York, 1961, $6.00.

A collection of papers, most of which have been presented at the meetings of the American Society of Criminology.

The chapter by Peter P. Lejins of the Department of Sociology of the University of Maryland discusses current theories about the influence of heredity on criminal behavior. The author reviews Lombroso's and Hooton's theories on the relation of anatomy to crime, concluding that they have had little impact on American criminology. The studies of Sheldon and the Gluecks, however, are receiving serious consideration.

Sheldon has showed a high correlation of criminality with the body type of the endomorphic mesomorph. The Gluecks also found a high percentage of mesomorphs to be delinquent. For the first time a plausible mechanism has been suggested linking heredity with environment. An endomorph is a man of action who acts to get out of a situation instead of just displaying general weakness. An ectomorph is not a person of action. Thus different types of physique may have different patterns of response which sometimes do, and sometimes do not, lead to criminal behavior. Only a rabid environmentalist could object to this hypothesis, according to Lejins.

It is pointed out that we are only in the beginning stages of the solution of the nature-nurture dilemma but that promising steps have been made in the area of criminology. The theory of the moment is at the same time a research assignment. This volume also discusses punishment, law enforcement, and sex, sanity, and society.

POPULATION GROWTH AND MANPOWER IN THE PHILIPPINES: A Joint Study by the United Nations and the Government of the Philippines. United Nations, Department of Economic and Social Affairs, New York, 1960, 66 pp. (paper).

There is much evidence for high fertility and a declining death rate in the Philippines, although the vital statistics for this country are very deficient. A birth rate of 46 per 1000 is indicated, but underreporting here is likely. If certain trends continue, the population could double in about 18 years. All projections point to a large population growth in the Philippines in the near future.

Fertility is only one of the subjects covered in this study which emphasizes the demography of manpower. It was undertaken not only to supply the government of the Philippines with demographic information for its planning programs but also to be of use to other countries.

A FAMILY PLANNING SERVICE IN RURAL PUERTO RICO: Christopher Tietze, M.D., Dattatraya N. Pai, Carl E. Taylor, M.D., Clarence J. Gamble, M.D. Publication No. 8, National Committee on Maternal Health, Inc., New York, 1961, 8 pp. (paper).

Reports on a fall in pregnancies resulting from a family-planning program in Puerto Rico from 1949-1958. Over a thousand couples from a rural area participated. Slightly more than half received instruction and supplies at home while the rest had clinic services only. Over 300 couples underwent surgery for sterilization.

Pregnancy rates of couples prior to the family-planning service was 83 per 100 years of exposure. During the time when five birth-control methods were used, it ranged from 28.3 to 42.3. Home visits were successful in encouraging the use of contraception. The condom and the diaphragm were preferred.

THE CURRENT STATUS OF FERTILITY CONTROL: Christopher Tietze, M.D. Publication No. 9, National Committee on Maternal Health, Inc., New York, 1960, 18 pp. (paper).

A useful summary by a world authority of methods used to control fertility and their effectiveness in the United States and other countries. The investigation in 1955, known as the Growth of American Families Study, is cited as the most comprehensive information on the extent of contraception in the United States. Some 70 per cent of the couples in this sample had been using birth control. Contraception was practiced by 75 per cent of the Protestant and 86 per cent of the Jewish wives, but only 57 per cent of the Roman Catholic wives used it. Education made a big difference, 84 per cent of college wives employing contraception as compared with 48 per cent of the wives who had not gone beyond grade school. A larger proportion of recently married couples in the United States use contraception than in the United Kingdom. American wives use the rhythm method more than the British, but the United Kingdom relies much more on coitus interruptus.

FILMS

A new film, The Diagnosis of Hidden Congenital Anomalies, is available free of charge

from the National Foundation, 800 Second Avenue, New York 17. It was prepared by Virginia Apgar of the National Foundation and L. Stanley Jones of the College of Physicians and Surgeons, Columbia University. It demonstrates to physicians and nurses a simple method for diagnosing certain anomalies shortly after birth.

OTHER PUBLICATIONS RECEIVED

Cold Spring Harbor Symposia on Quantitative Biology: Vol. XXV, Biological Clocks. The Biological Laboratory, Cold Spring Harbor, L. I., New York, 1960. Papers on the subject of biological clocks. Reflects the remarkable interest in biological clocks during the past ten years resulting from demonstrations that many animals navigate by the sun, using an internal chronometer.

A Symposium on Light and Life: Edited by William D. McElroy and Bentley Glass. Sponsored by the McCollum Pratt Institute of The Johns Hopkins University. The Johns Hopkins Press, Baltimore, 1961. Proceedings of the 10th McCollum Pratt Symposium held at The Johns Hopkins University in March 1960.

Elementary Human Physiology: Terence A. Rogers. John Wiley & Sons, Inc., New York, 1961, $6.50. A textbook in physiology.

Classics in Psychology: Edited by Thorne Shipley. Philosophical Library, New York, 1961, $20.00. A collection of excerpts.

Soviet Psychology — A Symposium: Philosophical Library, New York, 1961, $3.75. A discussion of the principles of the dominant schools of Soviet psychology and the theories underlying their educational programs by outstanding contemporary Russian psychologists.

Sociology of Crime: Edited by Joseph S. Roucek. Philosophical Library, New York, 1961, $10.00. The only publication in English which surveys broadly the sociology of crime, according to the editor.

The Predictive Process: Roy G. Francis. A Social Science Research Center Study, College of Social Sciences, University of Puerto Rico, Rio Piedras, Puerto Rico, 1960 (paper). The theory of prediction with special reference to Puerto Rico.

MEETINGS

Opening sessions of the 10th International Congress of the History of Science will be held at Cornell University and concluding sessions at the American Philosophical Society in Philadelphia. The Congress will extend from August 26 to September 2, 1961. Its president is Henry Guerlac of Cornell University. Inquiries should be addressed to the Secretary, 10th International Congress of the History of Science, Cornell University, Ithaca, New York.

The meetings of the biological societies which make up the American Institute of Biological Sciences are scheduled from August 27 to September 1, 1961, at Purdue University in Lafayette, Indiana. About 1,500 basic research papers, including genetics and evolution, will be given in programs arranged by 27 societies.

The Population Association of America will serve as host at the meeting of the International Union for the Scientific Study of Population which takes place at the Loeb Student Center, New York University, New York, from September 11-16, 1961. There will be sessions of interest to eugenics on factors affecting fertility in industrialized and preindustrialized countries, biological and medical aspects of mortality, and families and households. Further information may be obtained from Clyde V. Kiser, Milbank Memorial Fund, 40 Wall Street, New York 5.

The 50th Anniversary of the Family Service Association of America will be celebrated from November 12-17, 1961, at the Hotel Commodore in New York City. Key members of the Kennedy administration are expected to attend. A special session will be devoted to finding out what kind of families come for help to agencies.

More than 5,000 public health specialists from this country and abroad are expected to attend the 89th annual meeting of the American Public Health Association at

Cobo Hall in Detroit, Michigan, November 13-17, 1961. Joseph G. Molner, Health Commissioner of Detroit, is chairman of the local arrangements committee.

The fourth annual meeting of "The Society for the Scientific Study of Sex" will be held at 9:30 a.m., November 4, 1961, in the Barbizon Plaza Hotel, 106 Central Park South, New York City. The topic for the morning session is "Sex and Aging." Discussants are Dr. Harry Benjamin, Dr. Lissy F. Jarvik, Dr. Joseph T. Freeman, and Mrs. Donald Armstrong. Chairman is Dr. Hugo G. Beigel. The topic for the afternoon session is "Sex Factors in Schizophrenia" and discussants are Drs. Bernard C. Glueck, Jr., Jules D. Holzberg, Lothar B. Kalinowsky, and Sandor Rado. Chairman is Dr. Franz J. Kallmann.

HELEN HAMMONS

Periodical Reviews

GENETICS

ABO BLOOD GROUPS AND DISEASE

The association of blood group O with peptic ulcer was the subject of three recent papers in the *British Medical Journal.*

The ABO Blood Groups and Peptic Ulceration. E. W. Grahame. *Brit. Med. J.*, 5219 (1961), pp. 95-96.

ABO Blood Groups of Siblings of Duodenal Ulcer Patients. J. A. Buckwalter, R. E. Van Scoy, and L. E. Knowler. *Brit. Med. J.*, 5213 (1960), pp. 1643-1644.

Secretion of ABH Antigens in Peptic Ulceration and Gastric Carcinoma. E. Newman, G. S. Naifeh, J. E. Auer, and J. A. Buckwalter. *Brit. Med. J.*, 5219 (1961), pp. 92-95.

Grahame compared the blood group frequencies in a series of 1080 peptic ulcer patients, confirmed by laparotomy, with those expected on the basis of Fraser Roberts' data for 4540 blood donors from the same region in England. Blood group O occurred with significantly higher frequency than expected in the 722 duodenal ulcer cases and in the 126 patients with combined duodenal and gastric ulcer, but not in the remaining 232 persons with gastric ulcer alone. The decreased incidence of blood group A observed in duodenal ulcer patients was not significant, while the frequency of blood group B was significantly lower in both the duodenal and the gastric ulcer groups. The author concluded that there is a significant liability of persons with group O blood and a decreased risk for persons of group A to duodenal ulceration.

A different approach to this problem was employed on an Iowa population by Buckwalter, Van Scoy, and Knowler, who investigated 480 patients with duodenal ulcer and 1495 of their siblings. The siblings were divided into those with duodenal ulcer (80) and those not affected (1,186), and those whose health status was uncertain (229). The patients had the highest frequency of blood group O (55%), population controls (50,000 from various sources) the lowest (44.8%), and the siblings fell somewhere between the two extremes. Although the patients showed a significantly greater incidence of blood group O than did the population controls, the siblings with duodenal ulcer (affected) did not. As a matter of fact, blood group O appeared to be as frequent in the unaffected (47.2%) as in the affected (46.3%) siblings. No significant differences for group 0 could be demonstrated between the ulcer patients and any of their three sibling groups. As the authors point out, the frequency of blood group O in affected siblings was lower than expected not only in their own data but also in a previous study (Clarke *et al.*, *Brit. Med. J., 2* (1956), p. 725). Combination of the data from both these studies failed to provide an adequate number of sibships for definitive evaluation and for this reason the authors emphasized the need for further studies.

In the third paper, a possible mech-

anism for the relationship between ABO blood groups, peptic ulceration, and gastric carcinoma was evaluated by the Iowa group (Newman *et al.*). The authors suggested that the mucopolysaccharide ABH blood group substances secreted into the gastrointestinal tract protect the mucosa against ulcerogenic and carcinogenic agents. The population for this investigation included 479 verified cases of duodenal ulcer, 184 patients with gastric ulcer, 118 persons with gastric carcinoma and 1261 control subjects (hospital personnel and other patients).

A significant increase in nonsecretors of the ABH antigens was found for patients with duodenal ulcers compared with the controls, while no significant differences were obtained for patients with gastric carcinoma or gastric ulcers. No association of secretor status and severity of duodenal ulcer was found, leading the authors to suggest that secretion of ABH substances produces an initial protective barrier against ulceration.

CHROMOSOME STUDIES

Sex Determination: Diagnostic Methods. W. M. Davidson. *Brit. Med. J.*, 5217 (1960), pp. 1901-1906.

Intersex States. G. L. Foss. *Brit. Med. J.*, 5217 (1960), pp. 1907-1909.

In the first paper a general survey of the literature on sex chromatin and human chromosome investigations is provided together with current ideas about the development of normal and abnormal patterns of sex determination. While the second paper overlaps to some extent the presentation on the previous pages of the *British Medical Journal*, the clinical descriptions of the sex abnormalities are of value. The suggestion is made that all infants be screened to determine their nuclear chromatin pattern and that the chromosomes of questionable cases be studied in order to detect and treat as early as possible cases of abnormal sex development.

The Single-X Nature of Sex Chromatin in Man. S. Ohno and S. Makino. *Lancet*, 1 (1961), pp. 78-79.

Sex-Chromatin Body in Normal Human Testis. J. S. S. Stewart and A. R. Anderson. *Lancet*, 1 (1961), pp. 79-80.

Both articles indicate that the sex chromatin body in the normal female is derived from the heterochromatin of only one of the pair of X chromosomes. In general, the maximum number of sex chromatin bodies in somatic resting nuclei is one less than the number of X chromosomes. Stewart and Sanderson present an interesting formula for predicting the frequency of nuclei with one or more sex chromatin bodies in subjects with abnormal karyotypes.

Reproduction in Mongolism. H. Forssman, O. Lehmann, and T. Torsten. *Am. J. Ment. Defic.*, 65 (1961), pp. 495-498.

The authors review the literature and add one case of their own. As one affected mother-child case has been reported twice, there are now seven mongoloid mothers in the world literature, rather than the eight listed by the authors. These seven women have given birth to eight children (four normal, four affected). These data support the hypothesis that each child of an affected parent has a 50 per cent chance of receiving the abnormal chromosome.

Cytogenetic Studies in Chronic Myeloid Leukemia and Acute Leukemia Associated with Mongolism. I. M. Tough, W. M. Court-Brown, A. G. Baikie, K. E. Buckton, D. G. Harnden, P. A. Jacobs, M. J. King, and J. A. McBride. *Lancet*, 1 (1961), pp. 411-417.

The authors discuss an abnormality resulting from the deletion of material from chromosome 21. The resulting new small acrocentric chromosome, called Ph^1, because it was first reported in Philadelphia, has been observed in 13 out of 18 cases of chronic myeloid leukemia and in several cases of acute leukemia associated with mongolism. It is postulated that Ph^1 carries a locus concerned with leucopoiesis.

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ARTHUR FALEK
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POPULATION

Due to circumstances beyond control it was impossible to include reviews of Population in this issue. However, this section will be resumed in the September issue of the journal.

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TABLE 1

VALUES OF A, B, B/A AND 2B OBTAINED BY THE METHOD OF MORTON, CROW AND MULLER (1956) IN BRAZILIAN RURAL POPULATIONS.

Ethnic Groups	Abortions Plus Miscarriages			Stillbirths			Mortality After Birth			Total			
	A	B	B/A	A	B	B/A	A	B	B/A	A	B	B/A	2B
Caucasian	0.11	-0.23 ± 0.28	—2.09	0.04	0.15 ± 0.20	3.75	0.21	-0.01 ± 0.14	—0.05	0.36	-0.09 ± 0.37	—0.25	—0.18
Negro	0.09	3.06 ± 1.39	34.00	0.04	0.80 ± 0.85	20.00	0.23	0.51 ± 1.29	2.22	0.36	4.37 ± 2.08	12.14	8.74

ERRATUM

Table 1 of "Deleterious Mutations in Man," N. Freire-Maia, December 1960, EUGENICS QUARTERLY, 7(4):199-203, has been corrected to read as follows.

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